THE BOSTON
Medical and Surgical
JOURNAL

VOLUME 195 DECEMBER 23, 1926 NUMBER 26

NEW ENGLAND SURGICAL SOCIETY

THE OPERATIVE TREATMENT OF FRACTURES*

BY CHARLES L. SCUDDER, M.D., F.A.C.S.

GREAT attention is being focused today on a study of fracture problems in many large centres throughout this country. There is manifest also a very general interest in fractures in the profession at large. At this time I am attempting to state briefly certain facts bearing on the development of the operative treatment of fractures and to express a personal opinion as to the applicability of the operative treatment to special conditions.

When is the operative treatment of fractures of the long bones justifiable?

My reply briefly is: Whenever the operative treatment is needed or required to secure the best result following a given fracture.

It is unfortunate that today large groups of dependable and comparable case histories are not available for study and comparison.

I can recall watching with awe thirty-five years ago, Dr. John C. Warren, the attending surgeon in Ward twenty-eight at the Massachusetts General Hospital, score or slash a closed fracture of both bones of the leg for impending gangrene, due to tension from hemorrhage following damage to important vessels below the knee. The posterior and anterior tibial pulses were absent, the toes and foot were blue and cool. By these incisions Warren rendered a closed or simple fracture compound or open! The outcome of this logical and bold step was watched with apprehension. The man recovered with a sound leg.

Compare this episode with the certainty, assurance, and boldness accompanying a premeditated incision to the fractured bone today. The change in attitude toward such a procedure is almost unbelievable.

I can recall the arguments had at about that same time by C. B. Porter and J. C. Warren, master surgeons, over the use of absorbable vs. non-absorbable suture material in fracture of the patella. At that time, fracture of the patella was the only closed recent fracture treated occasionally by operation. I remember with what glee Dr. Warren heard of a refracture of a healed patelle sutured by Dr. Porter with silver wire! Of course, the refracture was thought to have been due to the use of the non-absorbable wire, and Dr. Warren considered at that time that he was more than justified in his contention for absorbable suture material!

Those were stirring times indeed!! Personal feeling ran high. We youngsters shared in the excitement and of course took sides with our chiefs. We discussed the problems at issue. Our chiefs nursed their temporary personal feelings engendered by differences in surgical technique and opinion. Those were days of military conformity in surgery. The climate of surgical opinion was foggy. The balanced and enlightened surgical mind of today was in the making.

What a contrast between then and now, timidity has given place to assurance, emergency measures to premeditated operations.

The operative treatment of fractures has come to its present important place as a recognized part of general surgery gradually, progressively, and comparatively recently.

Following the recognition in the sixteenth century of the importance of observation and the necessity of recording these observations, there came in the nineteenth century the influence upon surgery of anaesthesia, of antisepsis, of asepsis, and of the roentgen ray. Agencies each of fundamental basic importance.

In the nineteenth and twentieth centuries were taken the remarkable strides toward the perfection of general surgery, noticeably of neurological surgery, the surgery of the thoracic organs, abdominal surgery: including the surgery of the gastro-intestinal, genitourinary, and pelvic organs.

Diseases of all these various parts often killed the individual. The pathology of many of these diseases became generally known. Consequently the best minds of the time took cognizance of relief through appropriate surgery. "As is our pathology so is our practice." Osler.

It was thought that fractures of the skeleton *never killed*—they only crippled, deformed, and incapacitated. When, however, it came to be

*Delivered at the annual meeting of the New England Surgical Society, October 2, 1926.

recognized that fractures killed (and you recall that the mortality rate in compound fractures was from 85 to 90 per cent)! then attention was focused on the dire and lethal condition of an infected fracture. Through the work of Pasteur and Lister the mortality from compound fracture was greatly diminished. Today, an ordinary civil compound fracture has a low mortality rate.

The industrial awakening of the nineteenth and twentieth centuries, the great war, the appearance of complicated machinery, and the universal use of automobiles have been tremendous incentives to an improved treatment of fractures.

It is a stimulating thought and I believe a logical one that the serious discussion today of the operative treatment of fractures coincides with the technical perfection of pathological surgery. This is not a mere coincidence. General surgery has been improving, developing up to and for this event. It was impossible for the treatment of fractures adequately to develop until this perfection in pathological surgery had been attained. Lane's attempt at the promulgation of the operative treatment was unfortunate. Such ill-timed popularization was bound to be followed by terrible results and disasters such as have been seen in this country since 1909. Lives, limbs, the power to earn a livelihood were sacrificed in the popular attempts to improve fracture surgery by indiscriminate operating.

The non-operative methods *were* inadequate, as Mr. Lane pointed out. The logical direction for improvement at that time lay in a better non-operative treatment. Those times were not prepared for the general adoption of operative treatment.

The recognition of the functional importance of crippling due to fracture, an idea developed by Lane, has been growing for some years. The general industrial awakening to an exact economic value of this loss of function from fracture is a development of recent times.

That the limb is a part of an intelligent mechanism, that the will is involved in every fracture, that it influences the recovery of function: The significance of these facts is too little appreciated.

That bone is intimately associated with other tissues; that a fracture of bone is closely and complexly related to all parts of the body in ways as yet not entirely understood: these are comparatively modern facts and are bound to have their influence on the treatment of fractures.

That bone is a *delicate* tissue, that it demands and requires consideration because of its peculiar qualities is a new thought. That bone tissue must be handled gently in manipulation as well as at operation because of the influence upon the processess of repair of such trauma, is coming to be appreciated.

All these facts, together with the general perfection of the procedures of surgery should influence profoundly our appraisal of the operative method of treating fractures.

In this present consideration of the applicability of the operative treatment of fractures, I assume that the fractures are seen immediately following injury. They are recent fractures. There is a very large group of fractures which receive inadequate initial treatment. These cases seen from 1 to 3 weeks following injury—form a separate problem.

By the non-operative treatment, I understand that treatment which *honestly* employs:

1. The suspension of the limb;
2. Adequate traction;
3. Adequate counter traction;
4. A proper hold on the limb of the traction force;
5. Manipulation;
6. Pressure;
7. Counter pressure;

and secures the alignment of the limb, and brings about the movement of involved or contiguous joints as early as possible.

By the *operative treatment* I understand that method which approaches the seat of fracture through an incision. The fracture is directly treated.

I assume by the *operative* treatment

1. That the highest degree of safety to the individual will obtain;
2. That the surgeon and assistants are skilled in the use of the treatment;
3. That the surgeon possesses ability more than that needed for the ordinary care legally required;
4. That the surgeon has available necessary instruments and apparatus;
5. That the employement of that form of anaesthesia applicable to the case in hand, whether it be regional, local, spinal, or general, will be above criticism;
6. That the conduct of the operative treatment so far as the pre-operative and post-operative care are concerned is adequate.
7. I assume that the final and exact procedure chosen in the operative treatment of a given case is appropriate.
8. I assume that by this treatment the involved or contiguous joints are moved as early as is possible.

What are the *conditions* and the *facts* to be considered in formulating an opinion as to the applicability of the operative treatment thus conceived to fractures of the long bone?

1. A trial of the non-operative treatment

may be made for a few days; if it is then found to be unsatisfactory, the operative treatment may be used.

2. Skeletal traction will accomplish more than skin traction and by its efficiency may take the place of the open operation.

3. The number of open operations is being materially diminished by the increasing efficient use of skeletal traction.

4. The operative treatment may secure perfect reduction.

5. Absolute fixation may be had by operation.

6. Perfect reposition is desirable, for perfect reposition means more nearly perfect function.

7. Restoration of good form to a broken bone gives a chance of nine to one in favor of good function. Allowance of bad form will give a chance of two to one against a good result. (Hey-Groves.) This is not an accurate estimate but is suggestive.

8. The operative treatment is a safe treatment.

9. Infection is eliminated.

10. The dangers from the sequelae of sepsis are eliminated.

11. An uninterupted movement of joints is desirable after fracture. Absolute reduction and perfect fixation of the fracture fragments permit of the earliest movement.

12. The operative treatment will allow the maximum early movement in contiguous or involved joints. Early use of the damaged part stimulates the reparative processes.

13. Delayed union and non-union are not caused by direct treatment. I am not convinced that operation increases the likelihood of non-union.

14. Foreign non-absorbable materials are not always employed in the operative treatment.

15. Non-absorbable steel plates and screws and their equivalents are not irritating, if properly used.

16. Forcible manipulation required by the non-operative treatment may do actual harm and may be the occasion of delayed union.

17. Poorly conducted and poorly applied non-operative methods each may give poor results.

18. The results from a poorly conceived non-operative treatment should not be compared with the results from an ideal operative treatment.

19. Only similar kinds of fractures treated by well conducted methods should be compared.

20. The treatment will be modified to suit the social and industrial status of the patient.

21. Immediate, early reduction is desirable by both methods.

22. Operation is justifiable after trying non-operative treatment.

23. Operation is justifiable as an initial treatment in an increasing number of cases.

You all appreciate the difficulties attending a personal statement of this kind.

Fractures in children—the growing period.

The wonderful compensatory power of growth is depended upon to make up for the deficiencies of the non-operative treatment. How far should this "compensatory power" be permitted to influence the choice of primary treatment? It should not be permitted to influence the treatment of fractures in the lower extremity as it may be occasionally in the upper extremity.

In the upper extremity in childhood:

Certain fractures of the upper end of the humerus, high surgical neck fractures, I believe are best treated by operation and simple reposition, possibly without internal fixation. (Incision, digital confirmation, leverage, fluoroscopic reduction.)

In fracture of the external condyle of the humerus with displacement of the small fragment, the broken condyle should be accurately replaced by operation.

Certain irreducible supracondylar fractures of the humerus should be replaced by incision and direct leverage.

A few fractures of both bones of the forearm which cannot be reduced by gentle manipulation may well be reduced by incision and leverage. Fixation by a four-screw plate may be required.

Fractures of one bone of the forearm, irreducible by manipulation, may be well reduced by incision and leverage.

In the lower extremity in childhood:

Skeletal traction will replace many operations on the femur.

Most femur fractures can be satisfactorily reduced and held by non-operative methods.

Fractures in the adult. Upper extremity. Mobility is desirable.

Operation should be done upon

Fractures of the great tuberosity of the humerus with wide displacement;

Fracture of the high surgical neck of the humerus and dislocation of the head;

Irreducible high surgical neck fracture;

Transverse fracture at the middle of the shaft if apposition cannot be secured.

It is so difficult to be sure whether a musculospiral paralysis associated with a fracture of the humeral shaft is due to a divided musculospiral or to a contusion of the nerve; that is, it is so difficult to distinguish between a complete and a partial paralysis, that I am inclined to treat the fracture expectantly and delay

operation on the nerve. Having secured union of the bone, if the nerve is not permanently damaged, its recovery will be evident when the bone is united, and there will not have resulted undue loss of time in the repair of the nerve by the delay.

A T fracture into the elbow joint in an adult should be operated upon if the joint surface cannot be restored by position, and this is often impossible.

The neck and head of the radius should be operated upon when the resulting deformity from displacement will impair motion.

Operation should be done upon both bones of the forearm, if accurate alignment cannot be immediately obtained. If the radius or the ulna is fractured with displacement of the fragments, operative replacement is indicated.

Fracture of the olecranon is best treated by the open method.

Fractures in the adult. Lower extremity.

In the lower extremity stability is desirable.

The neck of the femur: At present there should be no primary operation on a fracture through the middle of the neck of the femur. When it is proved that the fracture is not uniting, operation may be done.

Shaft of the femur: In wage-earning laborers and in young adults there are many fractures of the upper, middle, and lower third that I prefer to operate on at once.

Fractures of one or both femoral condyles should be operated upon.

Spiral fractures of both bones of the leg are sometimes best operated upon.

Many fractures involving the ankle joint should be operated upon.

Skeletal traction in certain oblique femoral fractures will reduce and hold the displacement.

Final opinion as to the desirability of operation versus non-operation in any fracture must rest on accumulating facts. We have today but few facts from dependable sources. We have many impressions! The best returns in adult fractures of the shaft of the femur are the more or less comparable cases from the clinics of Sherman in Pittsburgh, Campbell of Memphis, Tennessee, and Conwell in Alabama. In fracture of the femoral shaft in husky young adults all these clinics are securing splendid results: one clinic by operation, steel plate and steel screws; one clinic by anaesthesia, traction, counter traction, manipulation, reposition, and plaster of Paris splint; the other clinic by skeletal traction and plaster fixation. The treatment by operation with plate and screws seems to give a little earlier return to work than do the other methods. An average return to work of from five to six months is a satisfactory achievement following fracture of the shaft of the femur.

My contention, and this is the hopeful aspect of the matter, is that we have two very efficient methods of treatment, each requiring further use, amplification, and careful recording. As the data accumulate, we shall get nearer and nearer to the propriety of stating at the outset, as to a certain fracture, this case should be operated upon at once; or, in this case the non-operative treatment will be satisfactory.

We may feel, I believe, that the operative treatment of fractures has come to occupy a definite and established place in general surgery. It is no longer a last resort in treatment. It is an initial method of choice. This is a very great advance in the conception of fracture treatment.

I should like to state here, and it seems to be appropriate to this subject, my conception of the qualifications of a surgeon for the operative treatment of fractures.

There are at least eight factors necessary to the qualification of a surgeon operating upon recent closed uncomplicated fractures:

1. An adequate knowledge of the patient, physically, mentally, socially, industrially.
2. An exact knowledge of the local conditions under consideration in the individual case.
3. Perfect instrumental, mechanical, and physical equipment including access to roentgen-ray apparatus.
4. An accustomed proved operative technique, delicately balanced.
5. An understanding of the sensitive nature of bone tissue, its reaction to injury, and the conditions influencing the process of repair.
6. A practical experience in the successful treatment of certain fractures by modern non-operative methods.
7. A knowledge of the various operative procedures for the immobilization of fractures.
8. An open mind in choosing the method best adapted to the case in hand.

A surgeon having the above qualifications is capable of safely treating fractures by operation.

A surgeon not having these qualifications should not employ the operative method in treating fractures.

The only underlying fundamental reason justifying the ideal operative treatment of a simple closed uncomplicated fracture is the probability of effecting the earlier return of the part to a more nearly normal function than can be secured by any other treatment.

It is established that in skilled and sane hands the operative treatment of certain recent closed fractures is a safe procedure, with an almost negligible mortality risk, with a high percentage of restored normal function in a reasonable time.

It is also established that in skilled and sane

hands the non-operative treatment of certain recent closed fractures is a safe and satisfactory method of treatment, securing a high percentage of restored normal function in a reasonable time.

It should be the aim of every surgeon of traumatism treating fractures to perfect himself in both these methods.

The one method demands as much training and skill as the other.

A complete knowledge of the advantages and the technical details of each method is essential to the highest efficiency.

A general surgical training combined with a special technical mechanical training affords the most nearly ideal preparation for the treating of fractures that is possible.

The way to the accomplishment of better treatment is in three directions:

Through undergraduate instruction;

Through graduate instruction;

Through lay propaganda.

A great constructive movement is on foot to secure better undergraduate medical student instruction. It is having its effect.

A great movement is already under way in the United States to enlighten and sympathetically help the general practitioner in his handling of fractures. This is being ideally carried out in Boston.

Some appropriate day there will be illuminating lay propaganda in fractures. Such propaganda will accomplish for fracture treatment somewhat the same result that already has been so well accomplished for cancer treatment. Such lay propaganda will provide the workman with the idea of where to find the best treatment and what he may reasonably expect from the treatment instituted in his behalf.

Perhaps the day will never come when the free choice by an injured workman of the physician he employs will be done away with, but the corporation and the insurance companies already recognize that they are together spending in this country and in this state millions of dollars for unsatisfactory initial treatment, resulting in long disability, and in long reconstructive treatment. Initial bad treatment is the occasion of most of the expensive reconstructive treatment.

Along with the above-mentioned movements which are really irresistible are:

The equally important research laboratory investigations on bone repair;

The focusing attention in every large and small hospital upon each fracture patient as a peculiar problem requiring skilled attention;

The discussion by interested surgeons, on fracture services throughout this country, of the various problems coming up in individual cases of fracture;

The exchange of experiences between surgeons in the friendly visiting clinics;

The direct and intimate association of the experienced and inexperienced men in the care of these cases, transmitting thus habits of thought and details of treatment, creating thus a climate of surgical opinion that will eventually and inevitably find its way to the remotest community.

From all these accumulated and accumulating experiences facts are being arranged in an orderly fashion, from which will eventually emerge further sane and efficient practical suggestions, valuable for the general practitioner and the surgeon.

Discussion of the Papers Read by Dr. Clark* and Dr. Scudder

Dr. Ernest A. Wells, Hartford: Dr. Clark's paper is unique in this that, whether the time of disability is long or short, the figures represent the exact length of time that his patients were disabled and these figures were compiled by an independent observer on the basis of the adjustments made with the injured parties under the Compensation Law of Massachusetts. We often hear surgeons say that by this or that method they get better results in that their patients return to work sooner than by some other method. But this is the first time that I have come across a paper where the exact figures for this item have been compiled in this independent fashion and for this reason I feel that the method should be particularly commended.

Dr. Kendall Emerson, Worcester: I think that this paper by Dr. Clark is a valuable contribution and fundamentally so. The Committee of the American College of Surgeons is attempting to duplicate this valuable contribution by appealing to the hospitals of the country and to individual surgeons whose names we have and are card indexed, and communications are being sent to these surgeons to secure such results. It is a very slow piece of work because it depends upon records, and records are very difficult things to get accurately, but we are trying to do it, and hope in time to accumulate evidence of this sort which will accentuate the information that has been presented here.

Dr. Stephen A. Mahoney, Holyoke: It seems to me that one fact in this last paper might attract our attention and that is the small number of days in minor fractures before the patient returned to work; and I would like to ask whether the patient simply returned to earning or to his former work.

Dr. W. I. Clark, Worcester: Returned to earning. In many cases the man cannot use the part that was injured in his old occupation, but

*Dr. Clark's paper appeared in the issue of December 2, 1926.

he uses it enough to earn wages although he has to favor it.

DR. STEPHEN A. MAHONEY: These are industrial figures?

DR. W. I. CLARK: Yes; this paper was written from the economic standpoint of work. They are figures from the engineering department which has charge of the compilation. I have nothing to do with it but to bring them together and analyze them.

DR. DAVID W. PARKER, Manchester. I would like to ask Dr. Scudder, does or does not cutting down on a fracture increase the convalescence time of that fracture, as a general proposition?

DR. STEPHEN A. MAHONEY, Holyoke: In regard to the paper of Dr. Clark the statistics are liable to be taken as a standard by insurance companies, and with the short duration of disability it looks to me that the paper should be carried out much more in detail because in industrial cases we see fractures of the foot lasting a year, and here we see fractures lasting only a month; and I should like to ask Dr. Clark if the man is taken back to the factory when he is able to do any kind of work, for nowadays we find that a man isn't usually taken back unless he can go back to the same job that he had before he was injured, which means a full day's work. If a man is sent back to any kind of work he is able to do, making the work fit the accident, it is much different from what the industrial accident board will demand or give back to the man injured. If he can't do the full day's work, he isn't taken back. So really it doesn't represent the conditions as they are in Massachusetts today with the insurance companies.

DR. ROBERT J. GRAVES, Concord, N. H.: I would like to ask the psychology of his method of getting these men back to work. I don't know how to do it.

DR. CHARLES L. SCUDDER, Boston: In reply to the question in regard to the incision increasing the time of convalescence that is a difficult question to answer in general without putting the question in a specific form. In general I believe that if the operation is wisely chosen that the convalescence time will be much less than if the incision were not used. Consider a fracture of the femur where traction and countertraction is used, ordinarily union with good alignment will result. If that same case were incised and the bones brought together there is slight delay in the healing process caused by the trauma of the incision even under ideal conditions; but the convalescence time will be diminished because exact reposition of the bones will permit early functional use of the joint.

DR. W. I. CLARK, Worcester: It is difficult to analyze the question from the Compensation Act. The Compensation Act in Massachusetts reads this way: that a man (quoting Act) is paid $16.00 a week until he is able to return to work, and when he returns he receives two-thirds of the difference between what he earns on his return and what he earned before.

Nobody expects these figures to be those of an earning member of the community, and nobody expects the man to return to the original work he was doing except where a man, for instance, had an injury to his arm who is a watchman. Therefore it is pretty difficult to answer the doctor's question. I wish he would put that again. You want to know why the time is so short?

DR. STEPHEN A. MAHONEY: I wanted to know whether the man was returned to his original job or to part time work and what the time and expense was based on.

DR. W. I. CLARK: He is given a job he is able to do; but the cost figure is figured on his original work.

DR. STEPHEN A. MAHONEY: But the insurance companies are not slow to take up what appears in medical papers, and there may be a mis-interpretation—that a man ought to return to work much earlier according to these statistics than they usually do.

DR. W. I. CLARK: In some cases we are ahead of the average time and in some cases we are behind. In the femur cases we are behind, as 378 days is a very slow time. I would be glad to talk on this in detail.

In regard to getting the men back to work, it is a training in co-operation and it is a trick you have to work out. I have been at it for 15 years. Confidence in your patient that you are not going to give the patient something he will not be able to do; and the second thing is that you are going to continue treatment while he is working, until he is back at his old work. I think the fact that they are getting a little baking or massage each day encourages them to come back and continue at their work more than anything else.

REMARKS ON THE HISTORY OF THE OLD SURGICAL AMPHITHEATRE*

BY FREDERIC A. WASHBURN
Director of the Massachusetts General Hospital

Mr. President and Members of the New England Surgical Society: It is very appropriate that this Association of the surgeons of New England should meet at the oldest hospital in New England and in the oldest hospital operating room in New England, the room in which we now are. On July 4, 1818, the corner stone of this hospital was laid in Prince's Pasture, at the extreme west end of Boston, with full Masonic ceremonies, in the presence of the Governor, the Lieutenant-Governor of the Commonwealth, the Selectmen of the Town of Boston, and a great concourse of citizens. The records show that this pasture was open to the bay on the west and looking to the south and west one could see the beautiful hills that surround Boston. The hospital was then built on the edge of the bay at the extreme western edge of the Town of Boston. Charles Bulfinch was the architect. The Trustees of the hospital were very fortunate to secure his services. He was the foremost architect of his period. He designed such buildings as the State Houses in Boston and Hartford, Connecticut, many fine churches and he collaborated on the Capitol at Washington.

During the last two years this Bulfinch Building has been made fireproof and all its facilities for the care of patients have been brought up to modern standards. Research laboratories and other useful adjuncts of the modern hospital have been installed. This old operating room has been restored to its original appearance, with the exception of the seats. The money for installing fireproof seats, which copy the original ones, was not forthcoming and so temporary seats have been installed. Otherwise, the room presents much the same appearance as it did when used as an operating theatre. The old seats rose higher than those in which you sit, they were in the form of a semi-circle with a pit where the operations were done. Imagine, if you will, old Dr. John C. Warren standing where I stand now, Surgeon in Chief, short in stature, austere, puritanical, a man of ability, with the best surgical training of his day, with initiative and force. He wore a frock coat and a stock. He had a special frock coat which he wore when operating. The surgeons in those days were proud of their operating coats. If such a coat was stiff with blood and pus so that it would almost stand alone, it was the source of great pride to its owner. In an operation if they wanted a ligature or a suture, they would take it from a pin cushion hanging on the end of the seats, with, of course, no thought of sterilization. These pin cushions exist now and may be seen in the instrument cases in this room, together with other interesting mementos of the surgery of former days. On the edge of the seats, in addition to these pin cushions, you would find the rope and the pulley ready for the reduction of dislocated hips. This, of course, was in the days before Dr. Henry J. Bigelow discovered the Y ligament and the method of reducing a dislocated femur by manipulation.

On September 3, 1821, the first patient was admitted to the hospital. Dr. John C. Warren was the first surgeon and James Jackson the first physician. They kept these positions for many years. In March, 1822, six months after the hospital was opened, Dr. Warren made a report to the Trustees of the hospital upon the surgical cases:

"Since the Hospital has been opened, for the admission of patients, three capital operations have been performed there and four of inferior importance.

"Of the former description, the first was the operation for Lithotomy. The patient recovered, and has returned in safety to his friends.

"The second was the operation for Popliteal Aneurism. The disease consists in an enlargement of the great artery of the ham, attended with violent pain; and if left to itself, it ultimately proves fatal, either by the bursting of the diseased artery, or the mortification of the limb. The subject was an industrious female belonging to Boston. The operation for this disease was performed soon after the patient entered the Hospital. The event was favorable, and she has since been able to resume her former occupations.

"The third capital operation was rendered necessary in consequence of an accident. This patient had his leg crushed by the fall of a cart. His constitution was impaired by the use of ardent spirits, and when he was brought to the Hospital, he was in a state of delirium. The limb mortified, the broken bones were exposed, and after various efforts to relieve him without removing the limb, it was thought necessary to take the opinion of the Consulting Physicians. These gentlemen agreed that the prospect of saving the patient's life was extremely small; but that it would be impossible to save the limb. In order to afford him some chance of recovery, they advised the amputation of the part. This operation was performed; and for some days the appearance of the patient was improved; but being upwards of 60 years old and with a broken constitution, in about a week or ten days after he sunk from debility."

These were the three major operations. Of the minor operations there were four, one for Prolapsus Ani, one for Fistula, one for the relief of Phymosis, and the fourth the extraction of a Wen from the neck.

Only seven operations in six months. I think that this is probably a fair description of what surgery was before the days of anaesthesia. It was surgery of the extremities or superficial surgery of other parts of the body.

*Delivered at the annual meeting of the New England Surgical Society, October 2, 1926.

Dr. John C. Warren was surgeon from 1821 to 1856. Other surgeons of that period were Drs. Hayward, Townsend, and later Drs. J. Mason Warren, Henry J. Bigelow, and Samuel Parkman. The name of Oliver Wendell Holmes appears as a Consulting Surgeon in this period.

Now we come to the great event with which this room is associated and that is the operation which you see described in the legend upon the wall before you:

"On October 16, 1846, in this room, then the operating theatre of the hospital, was given the first public demonsrtation of anaesthesia to the extent of producing insensibility to pain during a serious surgical operation. Sulphuric ether was administered by William Thomas Green Morton, a Boston dentist. The patient was Gilbert Abbott. The operation was the removal of a tumor under the jaw. The surgeon was John Collins Warren. The patient declared that he had felt no pain during the operation and was discharged well December 7. Knowledge of this discovery spread from this room throughout the civilized world and a new era for surgery began."

Like all great discoveries, the question as to who should receive the credit for surgical anaesthesia is bitterly argued. I quote from an address by Dr. John C. Warren before the Surgical Section of the American Medical Association on June 7, 1921:

"A decision has recently been reached by the Senate of the University of New York, based on recommendations from distinguished representatives of its electoral board from every state in the country, giving Dr. William T. G. Morton a place in the Hall of Fame.

"It is true that other individuals experimented also with ether, but not to a convincing degree, and therefore failed to affect in any way the surgical practice of the time.

"On October 16, 1846, Dr. Morton administered ether to a patient for a major operation in surgery at the Massachusetts General Hospital. The experiment was so successful that it was followed immediately by other operations on following days. To Dr. Henry J. Bigelow is due the credit of pointing out that this experience showed that ether was 'safe, certain, and complete.' It was a demonstration of the practicability of surgical anaesthesia which had been dreamt about for many years, and it was this triple feat that constituted the 'Discovery' and entitled October 16, 1846, to have a special name like that of our national holiday."

The news of successfully accomplished anaesthesia in a major surgical operation spread quickly throughout the world. As early as December 21, 1846, the first major operation in England under anaesthesia was performed by Robert Liston, at the old University College Hospital, London, who shouted:

"Hurrah! Rejoice! Mesmerism and its professors have met with a heavy blow and great discouragement. An American dentist has used the inhalation of ether to destroy sensation in his operations and the plan has succeeded in the hands of Warren, Hayward and others, in Boston. In six months no operation will be performed without this previous preparation. Rejoice!"

Others beside Drs. Morton and Warren are entitled to credit in the introduction of surgical anaesthesia. You will find, if you go to Washington, that the State of Georgia has put in the Capitol there, as each state is entitled to do, a statue to Crawford W. Long. Dr. Long was a respectable practitioner of medicine in Georgia. In 1842 as he had observed that people in an ether frolic barked their shins without feeling pain, he gave ether to a few patients for minor surgical operations. Unfortunately, he did not publish an account of this until 1849 and his description is vague and not convincing that anything further than primary anaesthesia was given.

Dr. Horace Wells, a dentist of Hartford, Connecticut, gave nitrous oxide gas for the extraction of teeth as early as 1844. Dr. Wells came to Boston in January, 1845, and was given an opportunity by Dr. Warren to demonstrate his method before students. This demonstration was a fiasco and Dr. Wells gave up the use of nitrous oxide gas and four years later committed suicide.

A bitter contest was waged in Boston for years as to whether or not the credit of the successful operations at the Massachusetts General Hospital was due to Dr. Morton or to Charles T. Jackson, a learned chemist and geologist. Undoubtedly Dr. Morton received suggestions from Jackson, but the consensus of opinion of the Hospital Staff and Trustees at the time and since has been that it was Morton's knowledge, Morton's originality and courage, together with the wise daring of Dr. John C. Warren, which made possible the satisfactory administration of surgical anaesthesia performed in this room October 16, 1846. Long, Wells, Jackson, and others are entitled to credit but their efforts made no impression upon the surgical practice of the time, whereas the work done by Dr. William T. G. Morton and Dr. John C. Warren, largely because of the stamp of authority placed upon it by the Massachusetts General Hospital, and its staff, revolutionized the surgical practice of the world.

To those of you who may wish to read on this subject in further detail, I would recommend the very complete address entitled, "A Consideration of the Introduction of Surgical Anaesthesia" given by Dr. William H. Welch, on the sixty-second anniversary of Ether Day at the Massachusetts General Hospital.

Pictures were then shown of the Bulfinch Building as it was originally, before the wings were added, of the Ether Room, old pictures of members of the staff, and daguerreotypes of early operations under ether, architects' drawings made in 1846, showing the laundry and kitchens of that time, pictures taken in 1856 showing the relation of the Harvard Medical School of that day to the Hospital, with water coming up to Hospital and School and the dissecting sheds built out over the flats.

ORIGINAL ARTICLES

THE INFLUENCE OF NEGATIVE PRESSURE IN THE SPHENOID ON THE OPTIC NERVE*

BY LEON E. WHITE, M.D.

In the study of optic nerve disturbances from focal infection, the one great stumbling block has been the difficulty of explaining why an optic neuritis should occur when the posterior sinuses were practically normal. The radiographs of these sinuses seldom show pathology. Their mucosa usually appears normal and even a microscopic study reveals but inconsequential changes. Yet the opening of these apparently normal sinuses benefits the neuritis. This clinical fact has been recognized by all rhinologists having experience with these cases and the literature is filled with reports of their brilliant results. The fact that such good results followed the opening of the sphenoids either alone or in conjunction with the ethmoids, has led to the conclusion that this procedure removed the focus of infection, and that the sinuses although appearing normal were yet in some mysterious way responsible for the eye condition. Many, therefore, advocated the opening of normal sinuses. Volumes were devoted to the investigation of the anatomical relationships between the sinuses and the optic nerve, and while in a way this work was valuable, it led to erroneous conclusions. It placed the adjacent sinuses too much in the limelight to the exclusion of more definite but distant foci. When the contra-lateral relationships of the sinuses to the optic nerve were described, it gave rise to extremely radical but unwarranted surgical procedures, such as bilateral, ethmoid and sphenoid exenterations for the relief of unilateral nerve disturbances. The fact that advocates of these radical procedures still exist leads me to discuss one phase of this subject that has not, as far as I know, been called to the attention of the profession. Even if it has been mentioned, it needs emphasis by repetition as it helps to explain why the opening of the posterior sinuses, even if normal, benefits optic neuritis. This brings us to the title of the paper,—"The Influence of Negative Pressure in the Sphenoid on the Optic Nerve."

It is a well recognized fact, as I have explained in several of my papers, that cases with optic neuritis usually have obstruction to ventilation and drainage of the posterior sinuses. This obstruction is usually produced by a middle turbinate wedged between the ethmoid wall and the septum. There is often a septal deflection to the affected side, although in some instances the septum may go to the opposite side and a compensatory hypertrophy of the middle turbinate takes place. Any of these conditions may so obstruct the normal openings of the posterior sinuses that negative pressure within them might readily ensue. This condition would probably favor the migration of the bacteria and toxins from the blood stream or lymphatics to the tissues adjacent to the optic nerve and a consequent neuritis might follow. This hypothesis would explain why we have a practically normal mucosa in most of these cases, as I could count on my fingers the sphenoids that appeared diseased among the many I have opened. The relief of the negative pressure and the depletion incident to the operation would naturally greatly benefit the neuritis. There are two phases of this theory of negative pressure that require elaboration,—1st, is it possible for this condition to exist within the posterior sinuses? 2nd, if it does exist, would this negative pressure favor the passage of the bacteria in the blood stream to the sheath of the nerve? I think it has been sufficiently proven by Billings and others that bacteria from most any focus may travel in the blood stream. As to the first phase,—the possibility of a vacuum condition of the sphenoid—let us consider similar conditions in the other accessory sinuses. There is, thanks to *Sluder's* investigations, a well recognized condition in the frontal sinus brought about by closure of the naso-frontal duct and known as vacuum disease of the frontal sinus. Sluder also described ethmoidal vacuum and considers it fairly common.

Brawley's description of the mechanism of negative pressure is so good that I will quote it rather than try to describe it myself. He says that "as a rule no history of nasal disease is obtainable. Close examination will reveal a swollen boggy turbinate or one which is anatomically so close to the lateral nasal wall as to press on the hiatus semilunaris and thus interfere with free interchange of air and free exit of normal secretion from the frontal and anterior ethmoid cells." He advocates resection of the middle turbinate to relieve this condition. "The etiology depends on the anatomic relationship of the middle turbinate to the hiatus semilunaris and the infundibulum. The air in the frontal and anterior ethmoid cells is imprisoned there by pressure of the middle turbinate against the lateral nasal wall, the turgescent tissues filling in the hiatus semilunaris and effectually blocking it. The lining mucous membrane of the sinuses absorbs the oxygen in the

*Read before the American Laryngological Association in Montreal on June 4, 1926.

imprisoned air and thus creates a vacuum. This absorption results in negative pressure and consequent swelling of the lining membrane with increased blood supply to this region. Stasis to a greater or less degree results. Pressure of the congested tissue on the contained nerve endings together with this stasis produces the symptoms and results in reflex vaso-motor disturbances in the circulation of the neighboring structures."

Negative pressure of the antrum largely due to the painstaking investigation of *Lynch*, is now well recognized. He states that "the condition is the production of a vacuum in the antrum not depending upon a previous recent inflammation in that cavity, and not associated with any marked nasal lesion, such as active suppuration, polypi or new formation. Truly a non-suppurative condition and a non-inflammatory one, there being no evidences of any inflammatory state in the nose, simply a condition of hyperemia, produced by the negative pressure, and being entirely relieved as soon as complete ventilation of the cavity is established. . . . Granting then that this antral cavity is closed by some process in the soft tissues or bone, we have many examples to prove that the confined air will be absorbed in this cavity as it is in the ear. It is a common experience for the general surgeon to meet with a localized emphysema, and to see the air in the tissues gradually absorbed. . . . These absolute facts leave no doubt as to the probability of the existence of a vacuum in the antrum, if the only means of ventilation of the cavity, namely, through the natural or accessory opening, is closed by some pathologic change, and the confined air absorbed by the processes referred to above."

While this closure may be accomplished by an acute tissue reaction to some invading infection, and may thus account for the headache that accompanies these conditions, the cases that form the basis for this article are all of the chronic, long-standing type and it is upon them I wish to lay particular stress, for they are far more likely to be overlooked in a search for the explanation of the varied symptoms they present.

"Of the fourteen cases that have occurred in my practice in the last eighteen months," says *Lynch*, "I was unable to find any gross nasal lesion that would lead to an examination of the antrum. In two there was a malposition of the middle turbinate, with a high deflected septum; the two others were incident to an atrophic fetid rhinitis."

"It is possible," says Lynch, "that the hyperplastic changes described by Uffenorde and Skillern and others might easily extend sufficiently far down the naso-antral wall to involve the opening into the antral cavity, or produce some change in the lining membrane by extension through the fibro-bony wall, accounting for the closure of normal and accessory openings in this way."

These hyperplastic changes are also found about the sphenoidal ostium and might readily play an important role in negative pressure in the sphenoid. In a recent communication from Dr. Lynch in reply to my query as to his views on negative pressure in the sphenoid, he states:—"I have seen four cases of the sphenoid in all of which after opening, all symptoms disappeared."

The middle ear, however, seems to give us our strongest proof, for in this accessory sinus, if you will permit me to so designate it, we have a drum membrane through which we can see and study negative pressure. It is well known that closure of the eustachian tube is shortly followed by absorption of the air within the typanum and a partial vacuum results. The ear drum is retracted and the patient is conscious of something a little out of the ordinary in the ear. Should there be complete closure of the tube for some days, the ear becomes filled with serum and we have what is known as a secretory ear. Reasoning from analogy we thus have a well recognized condition of negative pressure in the frontal sinus, the antrum, ethmoids and middle ear, all brought about by closure of the ducts or ostia, swelling of the mucosa about the nasal openings of these structures often being sufficient to bring about this condition. It is not necessary in these cases to have an acute infection, although it occurs frequently when there is one. This fact is equally true in cases with optic nerve disturbances. In most instances some previous nasal infection antedates the optic neuritis. Reasoning from this it would seem that closure of the sphenoidal ostium would produce negative pressure even if the sphenoid itself was not actually involved.

While it is probable that this vacuum theory has been greatly overworked and that many obscure neuralgias have been erroneously attributed to negative pressure, there are, without doubt, a certain number of true cases. Many patients suffer from occipital headaches of obscure origin. Is it not possible that negative pressure in the sphenoid may account for some of these? I do not wish to over-estimate its importance for I too well know the tendency to do this with every new idea. It is probably better at present to let it remain simply as a theoretical possibility.

As to the second phase of this subject,—Does negative pressure favor the neuritis? Here again we must reason by analogy. There are no large nerve trunks in close relationship to the frontal. The supraorbitals, however, do become sensitive to pressure. In the middle ear we again have our best evidence, for coursing along its inner and posterior wall is the facial nerve

enclosed like the optic within a bony canal, and, like the optic, separated from its adjacent sinus by the thin bony wall of the Fallopian canal. In closure of the eustachian tube there may be an exudate of serum. If this ear is ventilated by inflation or incision of the drum membrane, the mucosa quickly returns to normal.

Associated with an acute secretory ear there is occasionally an involvement of the facial nerve known as Bell's palsy. In most instances this palsy comes on in conjunction with an acute secretory middle ear. The negative pressure within the tympanum seems to favor the migration of bacteria from the blood stream to the sheath of the nerve. Reik claims that in all cases incision of the tympanic membrane, i. e., ventilation of the middle ear, hastens recovery. Of course we all know of many instances in which spontaneous recovery of the facial palsy occurs similar to the recovery of a retrobulbar neuritis. In both instances we have a neuritis causing in the one facial paralysis and in the other blindness. The facial nerve is not a highly organized structure like the optic and regenerates even after several months, while optic atrophy not infrequently follows an optic neuritis of short duration.

The etiology of Bell's palsy has been under considerable controversy like that of optic neuritis but cases have been reported by Dabney where rapid recovery followed the removal of some distant focus of infection. I have seen such cases following tonsillitis and I doubt not that were we more painstaking in searching for a focus it could usually be found. In the Bell's palsies spontaneous recovery being the almost universal rule, no great effort is made to investigate them. If bacteria from some distant focus reaches the facial nerve, if and as is usual this happens when there is negative pressure within the middle ear, does it not seem logical to conclude that a similar condition of negative pressure within the sphenoid favors migration of bacteria and toxins to the optic nerve? If this supposition is correct, it should exert an enormous influence on the method of handling cases of optic nerve involvement. If we will but admit that a closed sphenoid favors the invasion of the optic nerve by bacteria from the blood stream, the first and logical thing is to eliminate the focus which feeds these bacteria to the blood. The elimination of this focus should be followed by a subsidence of the neuritis and a return of the vision. In cases recovering spontaneously we probably have a transitory focus, acute rhinitis, tonsillitis, etc. When there is a constant focus chronic retrobulbar neuritis and atrophy follows.

The treatment of these cases may, for convenience, be considered under three headings:—

1st: Probably 50% of the cases would recover spontaneously. To submit these to any serious operative procedure not definitely indicated by the pathology present would be not only unnecessary but needless and meddlesome interference. When some definite focus is found as infected teeth or tonsils, removal is indicated irrespective of the eye trouble. When no definite focus is discovered local treatment by astringent sprays and ointments is sufficient.

2nd: In about 30% of the cases there is in addition to some definite focus of infection, marked nasal blocking by a deflection of the septum or hypertrophy of the middle turbinate. These cases are usually of moderate severity and there is considerable risk in waiting for spontaneous recovery. Even should this take place recurrences are common. Owing to the nasal deformity astringent sprays do not reach the sphenoidal ostium so that a resection of the septum or turbinectomy, or both, are indicated, in addition to the removal of the infecting focus.

3rd: There remains a small number of cases, probably less than 20%, where optic atrophy is to be feared. These are the cases which arise from an especially virulent strain of bacteria, or where through some anatomical peculiarity as a small optic canal, the nerve becomes subjected to unusual pressure. Here all one's skill both in diagnosis and treatment must be exercised. Procrastination spells failure. In these a definite focus must be sought and eliminated if found, but above all else early surgical ventilation of the posterior sinuses is demanded. By this is meant the removal of the middle turbinate, and the free opening of the sphenoid and posterior ethmoid cell. The other ethmoids should not be disturbed unless they show definite infection. It is at this point that many rhinologists become unduly alarmed and resort to unnecessary mutilation of the intranasal structures. I do not believe there is any more indication for a complete ethmoid exenteration than there would be for a mastoidectomy in the ordinary case of Bell's palsy, and a bilateral ethmoid and sphenoid exenteration would be as senseless as a radical mastoid for this transitory paralysis.

While I have found several sphenoids with thickening of the nasal mucosa on their anterior wall and considerable edema of the lining membrane, and believe this condition common in cases with optic nerve lesions, I have but one case where I could definitely state that the ostium was closed. This fortunately was in one of our confreres, Dr. J. L. Shipley of the Navy. He has written his own history and it so well describes the symptoms we would expect with negative pressure in the sphenoid that I am giving it as he wrote it four years ago. I made a most thorough examination of the anterior face of the right sphenoid and felt positive that the ostium had been obliterated by scar tissue

following the previous opening of that sinus. On removal of the outer wall of the sphenoid the sinus was found to be filled with a soft mass of tissue, probably edematous mucous membrane. It gave way readily under pressure with a cotton swab. This tissue was not disturbed. The swelling subsided rapidly and within two weeks was normal. The eye disturbance also rapidly improved. The history was as follows:—

I have been fairly subject to "colds" most of my life. My nose first required special treatment in November and December, 1908. A physician in Fayetteville. Arkansas, treated me for two months or more and made four separate cauterizations of turbinates. After this treatment I got along fairly well. However, in 1910, Dr. Moulton of Fort Smith, Arkansas, made one examination, cauterized one point lightly, and prescribed local treatments, i. e., salt water irrigations, which I used off and on for about two years. I still caught cold easily and a discharge persisted for a considerable time afterward.

From 1911-14 I was in England and all catarrhal symptoms cleared up.

In 1916 I was troubled with obstructions again and was treated by Dr. H. W. Loeb of St. Louis. Finally he performed a submucous resection. One side, I do not remember which, still gave trouble and Dr. James Smith of St. Louis resected a portion of the soft tissue of the lower turbinate. For several years then I had, as a majority of my patients in the service put it, "no more trouble with colds, and the like, than anybody else."

I was first troubled with eye strain in 1910. No error of refraction was found but as I was using my eyes a great deal for close work, a weak plus sphere was given.

During the summer of 1918 I was very subject to colds, and the central vision in the left eye became slightly hazy and a small scintillating scotoma was present. Examination of eyes by Dr. G. B. Trible of Washington, D. C., was negative for pathology, vod 20/10, vos 20/30 direct vision, peripheral vision 20/15. Up to this time vision in both eyes had been 20/10. After a time, scintillation disappeared, leaving a central scotoma which at 20 feet obliterated a 20/20 letter. There was, and is, also slight macropsia.

About July 19, 1920, while in St. Louis, a sharp attack of iritis developed in left eye; as I remember, there was no antecedent cold or other apparent cause. Vision dropped to counting fingers at 10 feet. There were deposits on Descemet's membrane and temperature of 101° for several days. Physical examination by Dr. Charles H. Nielson was negative, except for chronic constipation found present due to spastic colon. Spasticity was shown by X-ray plates and fluoroscopic examination. X-rays of teeth, sinuses and mastoids by Dr. Homer Whelon were negative. Blood, urine, Wassermann, were normal (National Pathological Laboratory).

Repeated nasal examinations by Dr. W. M. C. Bryan showed slight discharge from posterior ethmoids and possibly sphenoids. It is worthy of note that up to this time there had been no mention of sinus infection. Vigorous local nasal treatment was received. Complete dilatation of the iris was finally obtained and recovery was complete and unusually rapid.

About August 27, 1920, I had a slight cold, but with marked occipital headache. A night or so later I noticed that I coud not read the theatre program with the lights turned out in the auditorium, as I had been able to do up to that time. Next day there was a slight haze over the central vision of right eye, vod 20/15, and perhaps a slight enlargement of the scotoma in the left. I believe there was no scintillation. August 30, Dr. W. M. C. Bryan exenterated ethmoids, right and left, and opened both sphenoids. The lining mucosa of both sphenoids, but especially the right, was oedematous. This oedema and all eye symptoms subsided promptly. Vision in right eye returned to 20/10 in good illumination, though a slight but definite haziness of central vision remained in dim light.

The operation caused sharp ethmoidal and sphenoidal headache and I realized that this was merely an accentuation of a dull feeling between the eyes and at the occiput, which I had had constantly for a long time without being conscious of it. This feeling cleared up and I then remained essentially free from symptoms for nearly two years.

During the spring of this year, 1922, the right nostril became obstructed rather frequently and the rght eustachian tube closed up quite occasionally. About March 22, 1922, I had a cold with definite symptoms of sphenoidal irritation on the right side, i. e., occipital headache, more on right side, and marked nervousness. A scintillating central scotoma appeared in right eye. Vision dropped from 20/10 to 20/15. Examination of eye and nose by Dr. Stevenson of Newport, Dr. Richards and associates of Fall River, and eye examination by Dr. Verhoeff, were all negative for objective pathology. Dr. White found the right sphenoid to be closed. Forty-eight hours after he opened the sphenoid on April 22, all scintillation had disappeared, right nostril no longer became obstructed and the right eustachian tube ceased to give trouble. In thirty days vod was 20/10.

At present, October 18, 1922, there is a very faint haze over central vision of right eye in dim light. Vision is 20/10 and I am essentially free from nasal symptoms. I still catch cold rather easily but symptoms are usually mild and respond easily to treatment.

I doubt if it will ever be possible to definitely diagnose many cases of negative pressure in the posterior sinuses. The difficulty of accomplishing this is due to their remoteness. The floor of the frontal, the anterior wall of the antrum, and the thin orbital plate of the ethmoids, can all be palpated and a tentative diagnosis at least made. The transparent drum membrane permits inspection of the middle ear but in the sphenoid the most that can be expected is to discover sufficient obstruction or swelling to render this condition possible.

I have in my records about sixty unreported cases with some type of optic nerve disturbance. A detailed report of thirty-three of these will be found in the Transactions of the American Laryngological Association for 1926. This report will, I believe, substantiate my contention that most cases recover by simply removing the focus of infection. The tonsils appeared to be the only focus in ten, the teeth in seven, and the antrum in one. Both teeth and tonsils appeared infected in three cases, while in five it was found in teeth, tonsils and antrum. In but three cases were the ethmoids involved and even in these there was also infected teeth. The posterior sinuses were opened in but five of these cases. A definite focus was found in twenty-nine while in three it was thought to be sys-

temic, diabetes in one, hyperthyroidism one, post-scarlatinal one. A focus was not discovered in one case. Its surgical elimination was followed by normal vision in fourteen, marked improvement in two, slight in three, none in two. In six refusal to have it removed was followed by no improvement in five, and normal vision in one. Three cases recovered under treatment while one showed marked improvement. A fifth case (the diabetic) did not improve.

Thus, teeth and tonsils appeared to be the foci in 70% of these cases, while the ethmoids were involved in less than 10%, so there would seem little excuse for longer considering ethmoids and sphenoids the all important factors in optic nerve disturbances. They rarely require exenteration. The important thing to do in rhinological surgery as in all other branches is to remove the focus.

SUMMARY

The fact that the opening of the posterior sinuses, although apparently normal, usually relieves optic neuritis, has led to the conclusion that these sinuses are in some mysterious way responsible for the eye condition.

The foci actually responsible for most of optic nerve disturbances have been found in the teeth and tonsils. The theory of negative pressure in the posterior sinuses would explain the beneficial results which follow the opening of normal sinuses.

Negative pressure is considered under two aspects:—

1. Is it possible for it to take place in the posterior sinuses?

2. If it does exist, would this negative pressure favor the passage of the bacteria in the blood stream to the nerve?

Negative pressure is a well recognized condition in the frontal sinus, antrum, anterior ethmoids and middle ear, and is brought about by a closure of their ostia or ducts, so that reasoning by analogy a similar condition should be brought about by closure of the sphenoidal ostium.

In Bell's palsy negative pressure within the tympanum seems to favor the migration of bacteria from some distant focus to the facial nerve, so that it seems logical to conclude that a similar condition of negative pressure within the sphenoid would favor migration of bacteria to the optic nerve. If it is conceded that a closed sphenoid may favor the invasion of the optic nerve by bacteria from the blood stream, the logical thing is to eliminate the focus that feeds the bacteria to the blood.

Treatment is considered under three heads:—

1st: About 50% of the cases will recover spontaneously. Remove any definite focus. If none is found, surgical intervention is contraindicated.

2nd: In 30% of the cases the relief of nasal blocking is indicated in addition to the removal of the focus.

3rd: In somewhat less than 20% of the cases it will be found necessary to open the posterior sinuses to prevent optic atrophy.

Thirty-three optic nerve cases are analyzed. In but 15% were the sphenoids and posterior ethmoids opened and even in some of these it was unnecessary. Post-operative cicatricial obliteration of the sphenoidal ostium with resulting negative pressure was found in one case.

BIBLIOGRAPHY

Billings: "Focal Infection." Published 1918.
Sluder, Greenfield: "Headaches and Eye Disorders of Nasal Origin." Published by C. V. Mosby Co., 1918.
Brawley, Frank E.: "Relation of Diseases of the Nasal Accessory Sinuses to Diseases of the Eye." Jour. A. M. A., Vol. 48, No. 12, 1907.
Lynch, R. C.: "Vacuum Diseases of the Maxillary Sinus." Annals O. R. and L., Vol. 23, 1914.
Reik, H. O.: "The Relationship Between Otitis Media (Non-suppurative) and Facial Paralysis of the Refrigeratory Type." Trans. Amer. Otol. Soc., 1904. Text-book on Diseases of the Ear, Nose and Throat. Published in 1911.
Dabney, Virginius: "Bell's Palsy: Four Cases of Infectious Origin." Trans. Amer. Acad. Ophthal. and Otol., 1924, p. 294.

COMPARATIVE EFFECTS OF BOROTARTRATE AND OF LUMINAL ON THE SEIZURES OF EPILEPSY*

BY WILLIAM G. LENNOX, M.D., AND L. H. WRIGHT, M.D.

ANY non sedative drug which holds forth promise of relieving the condition of those who are subject to recurring convulsions deserves careful trial. Borax is one of the numerous substances which has been advocated and used with indifferent success for many years. Recently Marie[1] and his associates have written hopefully of a preparation of boron, called potassium borotartrate. This has the formula CO_2H—CHOH—CHO—BO—CO_2K. Marie has published the results with the use of this drug in 10 cases of epilepsy. In this group the total number of seizures in the month before treatment was 310, and in the three following months, in which patients were each given three grams a day of the borotartrate, the number of seizures were 181, 118, and 89 respectively. Since the publication of these observations, a number of European clinicians have spoken fa-

*From the Department of Neuropathology, Harvard Medical School, and the Monson State Hospital. This paper is No. 52 of a series of studies in metabolism from the Harvard Medical School and allied hospitals. The expenses have been defrayed in part by a grant from the Proctor Fund of the Harvard Medical School for the study of chronic diseases. Grant has also been received from the Committee on Epilepsy, New York City.

vorably of results secured with this drug. Thus Bénard[2] obtained improvement in eight out of eleven cases. In these eight, seizures numbered 352 in the month before treatment, and 117, 91 and 69 in the three months during which potassium borotartrate was given. Trocello[3] found decreased frequency of spells in 8 cases. In the 10 patients successfully treated by Willemse[4], therapeutic results were obscured because luminal and boric acid were combined with the borotartrate given. Gardere, Gignoux and Barbier[5] reported decreased frequency of spells and increased intelligence in a single case. Krebelsberg[6] found the drug useful in twilight states. Cuneo[7], Neuberger[8], Lereboullet[9] and Laignel[10] recommended the use of borotartrate without presentation of supporting evidence. Liquor[11], Torres y Lopez[12], Prati[13], Grassi[14] and Regnard[15] have presented case reports.

Marie has little to say concerning the mechanism by which potassium or sodium borotartrate lessens convulsions. Bigwood[16] believes that beneficial result is due to the production of acidosis. He has measured the pH. of blood serum in half a dozen cases of epilepsy during treatment. From four to six grams a day of "borosodine" were given. In four of the cases charts showed diminished alkalinity of the blood. In two other cases alkalinity remained the same or was increased. In addition to this clinical trial, a dog was given 12 grams a day of "borosodine" for several days. After an interval of 10 days a single measurement of pH. was in the vicinity of 7.12. The situation is somewhat obscured by Bigwood's statement that patients were benefitted by "borosodine" even when pH. of the blood was not altered. However, because borotartrate is an acid forming salt, and because there is evidence that conditions which induce acidosis may have a favorable effect on the seizures in epilepsy, it would seem plausible that any beneficial effect of borotartrate would be due to the acidosis induced.

Marie[17] and Boitzi[18] have stated that some of the preparations of borotartrate on the market are not pure. We, therefore, secured medicine directly from Marie's druggist in Paris. Dr. Leonard W. Cretcher of the Mellon Institute of Industrial Research, University of Pittsburgh, was good enough to undertake examination of this material with reference to its acidity. He informed us that one gram of the potassium borotartrate when titrated with N/10 NaOH, using phenolphthalein as an indicator, was neutralized by 26 c.c. of the alkali. The addition of a few grams of mannite required 42 more c.c. of the NaOH to bring the solution again to neutrality. The gram of borotartrate had, therefore, the capacity of neutralizing 68 grams of alkali. Theoretically, one gram of pure potassium borotartarte in the presence of mannite will neutralize 93 c.c. of N/10 alkali. Dr. Cretcher prepared potassium borotartrate which was approximately pure. The patient who receives 3 grams of Dr. Cretcher's preparation gets the equivalent of 280 c.c. of N/10 acid. We are greatly indebted to Dr. Cretcher for supplying us with a sufficient amount of his borotartrate to make the clinical trial here recorded.

It is obvious that in the use of any drug for control of convulsions, one needs to know not only whether the use of such drug is attended with beneficial results, but also whether such results are better than can be obtained with a standard sedative, such as luminal. It is also important that a long period of observation should precede the trial, for with any group of patients there is great fluctuation in the number of seizures from month to month. Thus, the previously quoted observations of Marie[1] and his associates and of Bénard[2] may be criticized because their trial of borotartrate was preceded by a control period of only one month.

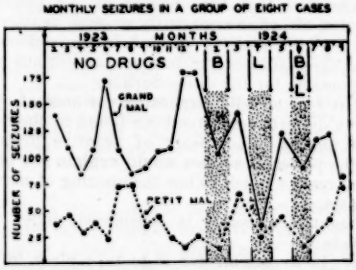


FIGURE 1. Comparative effect of potassium borotartrate and luminal on seizures. Ordinate represents the number of seizures per month. Abscissa represents months. Number of grand mal is shown by solid line, petit mal by dotted line. The letters indicate the months during which medication was given, as follows: B, potassium borotartrate, 3 gms. a day; L, luminal, 3 grains a day; B and L, both borotartrate and luminal in the above named doses.

We selected a group of eight patients with epilepsy who had been residents at the Monson State Hospital for at least a year. These patients for the first six months of the period recorded in Figure 1 had received only such drugs as their condition required. This meant the occasional use of sedatives at the time of a series of convulsions. During the next six months they received no sedative drugs whatever. It will be noted by the chart that the total number of monthly seizures in this group of eight patients fluctuated considerably. It is necessary to consider the records of subsequent months in the light of this preliminary year of observation. During the month of February, 1924, each patient received 15 grains of potassium borotartrate three times a day, approximately 3 grams a day. The next month no drugs were given. The following month the patients received a grain and a half of luminal twice a

day. No drugs were given the next month. The following month both luminal and borotartrate in the previously used dosage were given. Following this there was a period of three months in which medication was withheld. Inspection of the chart shows that seizures during each month of medication were somewhat less frequent than during subsequent drugless months. However, the only marked drop in number of convulsions during this period occurred in the month in which patients received luminal. During this time grand mal seizures were less than half their usual frequency. There was no commensurate diminution in the number of petit mal. Apparently this group of patients did not do as well on a combination of borotartrate and potassium borotartrate secured from Paris, and in much larger dosage than was employed by Marie; viz., 12 gms. a day. Occurrence of gastric symptoms prevented use of the drug for a longer period. Only a moderate dosage of luminal was used (1 c.g. or 1½ grains daily). Inspection of Figure 2 shows that during the eleven day period with borotartrate the frequency of seizures diminished to less than ten a day. Marie has stated that maximum effect of borotartrate is obtained only after prolonged use. In our patient, however, maximum effect was secured on the 4th day of use. Following the ingestion of luminal the seizures disappeared entirely. In a second trial of luminal continued for four weeks, seizures returned during the last

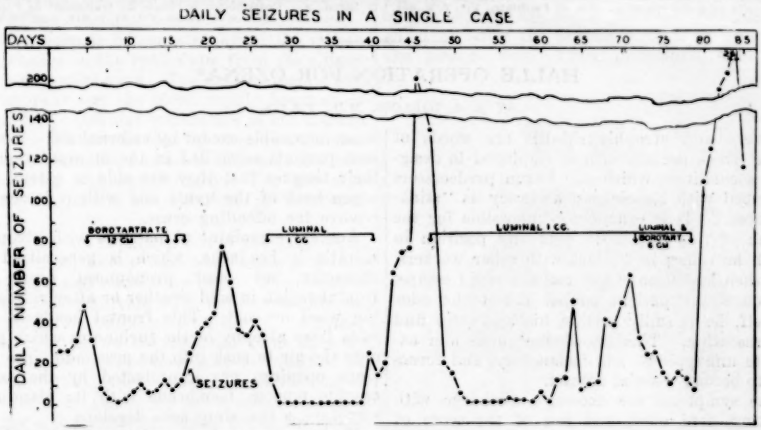


FIGURE 2. Effect of potassium borotartrate and luminal on the frequency of myoclonic seizures in a single case. Ordinate represents daily number of seizures; abscissa days. In order to save space the ordinate scale is deleted between 140 and 200. For this reason, the peaks of the curve are higher than they appear to be.

luminal as on luminal alone. It will be noted, however, that in the corresponding month (the sixth) of the previous year, seizures were unusually frequent. It is possible, therefore, that in this month the seasonal variation influenced results. Experiment with this small group of patients did not encourage longer trial with a larger group.

The patients included in this study, being chronic institutional cases, are the most difficult in which to secure therapeutic results. Better effect with borotartrate might be obtained from patients not requiring custodial care. We made trial on one such patient, who was being treated in the Nerve Department of the Boston City Hospital. She was having very frequent myoclonic seizures. Figure 2 shows the daily number of such seizures during periods in which she took potassium borotartrate, in contrast with two other periods in which she took luminal and a combination of borotartrate and luminal. With this patient we used the sixteen days. During the last eight days 6 gms. a day of borotartrate were given in addition to the luminal, with consequent reduction in the number of seizures. With both borotartrate and luminal there was great increase in frequency of seizures after medication was stopped. The daily average number of seizures during and after the use of each drug was approximately the same as the average number in the control period in which no medication was given. When used for these short periods both luminal and borotartrate acted merely as a dam. With the dam removed, the accumulated seizures manifested themselves.

A comparison of the effect of borotartrate and of other acid forming salts will be presented elsewhere.

CONCLUSIONS

In a group of nine epileptic patients beneficial results attended use of potassium boro-

tartrate but were less favorable than results obtained with luminal.

BIBLIOGRAPHY

1 Marie, P., Crouzon and Bouttier: Le tartrate borico potassique et la medication borée dans le traitement de l'epilepsie. Presse Medicale, Vol. 28, p. 713, 1920.

2 Bénard, A.: Le traitement de l'epilepsie par le tartrate boricopotassique. Thèse de Paris, Jouve & Co., pp. 1-48, 1920.

3 Trocello, E.: Il tartrato di borile e potassio nella cura dell'epilessia. Ann. di med. nav. e colon., Vol. 2, p. 665, 1921.

4 Willemse, A.: Die Behandlung der genuinen Epilepsie mit einer Kombination von Borpraparaten und Luminal. Nederlandsch tijdschr. v. Geneesk., Vol. 66, p. 2407, 1922.

5 Gardere, Gignoux and Barbier: La medication borée dans l'epilepsie—Resultats de 6 mois d'application. Jour. de med. de Lyons, Vol. 3, p. 79, 1922.

6 Krebelsberg, E.: Behandlung der Epilepsie mit tartarus Boroxatus. Psychiatr. Neurol. Wochen., Vol. 22, p. 389, 1921.

7 Cuneo, G.: Intorno al meccanismo di azione dei sali di boro nella cura dell'epilessia. Riv. di Palchiatr., Vol. 9, p. 381, 1921.

8 Neuberger, L.: Vues pratiques sur l'epilepsie. Rev. gêner. de clin. et de therap., Paris, Vol. 38, p. 313, 1924.

9 Lereboullet, P.: Traitement de l'epilepsie chez l'enfant. Bull. méd., Paris, Vol. 38, p. 1157, 1924.

10 Laignel, Lavastine and Voison: Traite de Pathologie medicale et de Therapeutique appliquée. A. Maloine & Sons, Paris, Vol. 8, p. 647, 1925.

11 Liquor: Medicamento contro l'epilessia a base di tartrato di borile e potassio. Rassegna di clin. terap., Roma, Vol. 20, p. 87, 1921.

12 Torres y Lopez, A. J.: Estudio del tratamiento de la epilepsia por el tartrato borico potasico. Med. ibera Madrid, Vol. 15, p. 519, 1921.

13 Prati, L.: Cura dell'epilessia coi tartrato boro-potassico. Rassigna di clin. terap. Roma, Vol. 21, p. 203, 1921.

14 Grassi, G. B.: Tartrato borico potassico nella cura dell'epilessia dell'infanzia. Pensioro med. Milano, Vol. 11, pp. 725-727, 1921.

15 Regnard, M.: Traitement des manifestations epileptiques par le phénylethyl-malonyluré associé au tartrate boricopotassique. Bull. gén. de thérap., Paris, 174, 585, 1923.

16 Bigwood, E. J.: L'Equilibre physico-chemique du sang sans l'epilepsie. Ann. de Medicine, Vol. 15, p. 119, 1924.

17 Marie, P., Crouzon and Bouttier: De la necessite de verifier la purete de tartrate borico-potassique dans son emploi contre epilepsie. Bull. Acad. de med., Paris, Vol. 84, p. 209, Nov., 1920.

18 Boltzi, A.: Contribution à l'étude du traitement de l'épilepsie. Rev. Med. de la Suisse, Vol. 45, p. 625, Aug. 25, 1925.

HALLE OPERATION FOR OZENA*

BY R. A. BARLOW, M.D., F.A.C.S.

OZENA and atrophic rhinitis are words of polite Greek derivation now employed to designate a condition which our Saxon predecessors described with Rabelaisian accuracy as "stinking nose." It is practically impossible for the victim of this disease to hold any position in which he comes in contact with other workers, for when he enters others make a rapid escape. Although the patient cannot detect the odor himself, he is sadly certain his associates find it nauseating. This knowledge cause him exquisite unhappiness and melancholy, and forces him to become a social outcast.

The symptoms are excessive discharge with crusting, foul odor, and loss of the sense of smell. The crusts vary in size from small, hard chunks to enormous casts of the entire nasal chamber which are removed with difficulty and are frequently associated with nasal hemorrhage. The patient develops his own cleansing technic, which is generally some sort of lavage with hawking and blowing.

Difficulty in breathing depends on the size of the crusts, as the turbinates become so atrophic that they have little or no part in the obstruction. As the mucous glands disappear and fail to moisten the surface of the larynx, hoarseness and cough develop. If the atrophy extends to the pharynx, huskiness and cough are more pronounced and the picture of pharyngitis sicca, as described in the various textbooks, is observed. The crust in this region often produces the syndrome of infection of the sphenoidal sinus, as it mechanically blocks the osteum of the sinus, thereby interfering with its drainage and ventilation. The lack of mobile tissue in the vault of the pharynx renders dislodgement of the crust impossible except by external aid. I have seen patients so skilled in the manipulation of their tongues that they are able to extend the organ back of the uvula and with it loosen or remove the offending crust.

Another complaint of patients with atrophic rhinitis is headache, which is generalized in character but most pronounced over the frontal region in cold weather or after riding in the wind or cold. This frontal headache results from atrophy of the turbinates which permits the air to rush into the nose, and since the sinus openings are unprotected by the usual erectile mucous membrane with its glandular structure, a real sinus ache develops.

Various causes of atrophic rhinitis have been suggested. One writer is persuaded that persons who live or work in moist air are prone to develop ozena, as he had observed several cases in laundry workers. Its origin has been attributed to acute exanthems, particularly measles and small pox. Even the accessory sinuses have been accused as irritating agents, and "familial tendency" has come in for its usual share of speculation. Perez isolated the coccobacillus, an acid fast organism, and grew it in bouillon broth, but the production of a vaccine therefrom did not prove beneficial. In 1915 we isolated the organism and experimented on rabbits. Crusts similar to those observed on the turbinates of patients were found accompanied by the characteristic odor. Nothing of importance was developed, however, and the work seemed of little value. Horn produced an autogenous vaccine and made encouraging reports, but other workers following the same technic were unable to secure the same results. A complete analysis of all theories regarding the cause of the disease brings us to one conclusion

*From the Section on Otolaryngology, Jackson Clinic, Madison, Wis.

which is substantiated by all the various investigators, namely: the exact etiology is unknown.

Atrophic rhinitis occurs very commonly in eastern Russia and central Europe. In this country it is observed in city dispensaries and clinics, but appears rather seldom in private practice. It is not contagious; one member of a family or group may be affected and others escape even the slightest tendency. It occurs both in males and females, and we have observed it at all ages from 3 to 68. Geographical location, climate, and altitude seem to have little influence on its incidence. Most of the cases are observed among poor persons, and it has occurred to me that the cause of the disease may be improper nutrition or some metabolic disturbance.

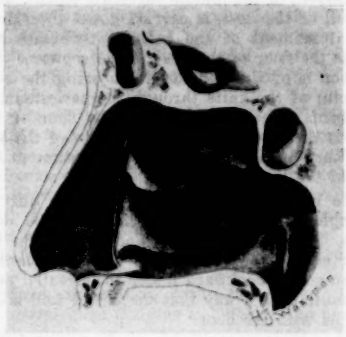
The disease is a true atrophy of the mucosa with change in the epithelium from the ciliated to the pavement type. The mucous glands tend to disappear and the blood supply becomes impoverished. Cellular infiltration is not marked, since the picture is that of chronic inflammation with atrophy, not acute inflammation with hyperplasia and hypertrophy. For the same reason polyps are not observed in this condition.

Examination reveals crusts of the nasal chamber, a thick, sticky secretion, anemic mucous membrane, and nauseating odor. There may be an ethmoid or antrum infection on one side, but the atrophic condition is bilateral. Furthermore, clearing up the infected sinus does not cure the ozena. The nose may be broad at the base and the bridge somewhat saddle shaped, but one must not preen one's self too soon on making a diagnosis by facies, for in the majority of these cases the Wassermann is negative.

The various cures are too numerous to mention. Enthusiasts have advocated almost every sort of a drug and chemical, including lactic acid, phenol in glycerine, iodin in glycerine, scarlet red ointment, ichthyol unguentum zinc oxide, and malachite green (Lynch). This last named drug produces a temporary edema which ameliorates the symptoms for the time being but soon exhausts itself. The crusting may be diminished by using 25 per cent glucose in glycerine. After the flakes are removed the nasal chamber is swabbed with the glucose-glycerine preparation. The glucose seems to have an inhibitory effect on the crust formation with consequent decrease in the odor; but this at best is only transient, its value depending upon the change of flora.

The surgical treatment of atrophic rhinitis has been obviously unsuccessful. In earlier days when intranasal surgery boasted only of turbinotomy and when little was known of nasal pathology the most common practice was to remove a turbinate,—thereby aggravating the existing condition. Later, rhinologists attacked the sinuses and for a while the literature was quite encouraging first with its recommendations of surgery of the ethmoid, then of the antrum. The surgeons, finding the sinuses occasionally involved, advanced the theory that sinusitis must be the causative factor. More recently we have come to regard the coexistent sinusitis as a part of the syndrome rather than as the cause of the disease.

The next step in the evolution of the surgical treatment of this condition rests on the theory that since the nasal space was abnormally large, it should be curtailed. Beck and others devised various procedures whereby paraffin wafers, cartilage, bone or even celluloid was inserted beneath the septal perichondrium in an effort to bulge the septum enough to diminish the passage way. These procedures were not



View of lateral nasal wall showing the opening into the antrum and the wall partially mobilized.

successful and our early hopes were again dashed on the rocks.

For a few years little was advanced in the treatment of atrophic rhinitis, and the rhinologist was none too effusive in his greeting of a patient with ozena. In 1917, Lautenschlager published a description of his operation for ozena which was the first step in what now seems to be a procedure of real benefit. He advanced the lateral wall of the nose toward the septum, denuded the antrum of its lining, and turned an oral mucosal flap into the cavity. This afforded relief, but, as Lorie very wisely observes, it was a "formidable procedure, requiring a rather mutilating operation, destroying, to some extent, normal tissue, requiring a general anesthetic and only to be performed in the hands of the most skillful."

It remained then for Max Halle to simplify the technic so that the operation can be performed under local anesthesia with most gratifying results. The so-called Halle operation

was introduced in this country by Doctors Lorie and Lux in 1922 and their first report on eighteen successful cases was published in 1924. Rhinologists have been slow to adopt it, but in our opinion it is the most satisfactory procedure as yet devised for the treatment of ozena.

With cocaine mud applied in the usual manner for intranasal anesthetics and a submucosal injection of novocaine, the work may be done with very little discomfort to the patient. A preliminary hypodermic of scopolamine, gr. 1/200, and morphine, gr. 1/6, may be given one-half hour before the operation. When the patient is prepared all crusts should be removed and the mucous membrane cleansed with a cotton swab saturated in dilute oil of lavender. This neutralizes somewhat the nauseating odor which is quite unpleasant even to surgeons.

The initial incison is started in front of the attachment of the middle turbinate on the lateral wall of the nose, is carried down anterior to the attachment of the inferior turbinate and across the floor of the nose to the septum. The incision is of sufficient depth to bring the edge or point of the knife through the periosteum to the bone. The periosteum of the floor of the nose is then elevated to the junction of the soft and hard palates and from the septum to the rise of the lateral wall.

The next step is to chisel into the antrum through the vertical incision in the lateral wall. Then, with the chisel flat and pointed in the lower end of the vertical cut the instrument is driven gently backward under the elevated periosteum in such a way that the antral wall is cut parallel with the floor.

This procedure results in an L shaped cut in the bony wall. Now, with the chisel inserted in the vertical cut and used as a lever, the lateral nasal wall is forcibly pried out until it hangs vertical in the nasal chamber. The turbinates, which have been previously scarified as have also corresponding areas on the septum, are in apposition to the septum. This permits a clean view of the antrum, which is carefully wiped out.

The antrum is then packed sufficiently tight with iodoform gauze to maintain the lateral nasal wall in contact with the nasal septum until adhesions form between the turbinate and septum. The initial packing is allowed to remain undisturbed for four or five days. Upon removal of the packing the antrum is cleansed and the upper angle of the dislocated lateral wall and the agger nasi is packed to keep the turbinates in contact with the septum. If the lateral wall has been sufficiently dislodged and mobilized, this is accomplished quite comfortably. The packing is replaced about every three days during a period of from three to five weeks, or until the adhesions are well formed and the granulations about fill the antrum. This results in marked diminution of the nasal chamber and if possible, the adhesions should not be disturbed for three or four months when they may be cut. The turbinates are then seen to extend into the nose and appear much healthier than before.

Both sides of the nose are operated on at one sitting. The results are most satisfactory from both the patient's and the rhinologist's standpoint. The patient must continue to care for the nose by douching, as the normal mucous membrane is never restored and there is bound to be some dropping of mucus. The offensive character of the discharge, however, disappears.

It is not definitely known in just what way the rejuvenation of the mucous membrane takes place. Lorie believes that the narrowing of the breathing space and the stimulation of blood supply from the septal adhesions are largely accountable. Certain it is that the mucous membrane appears moist, velvety and deeply colored. The secret of good results depends upon the after care; the lateral wall must be kept packed against the septum a sufficient length of time. If the patient can not or will not stay or coöperate the required time, the results will be of very little value. If contraction of granulation tissue tends to pull the nasal wall back toward the antrum in the first two weeks, it may be necessary to refracture the nasal wall against the septum.

Dr. Lux, who had personally observed many of Halle's cases more than three years postoperatively, reports that the results were brilliant. Lorie and Lux also report eighteen cases of more than two years' standing in which the outcome was equally satisfactory. We have performed Halle's operation on three patients at the Jackson Clinic during the past eighteen months with highly satisfactory results.

REFERENCES

Halle, M.: Die operative Therapie der Ozäna. Arch. f. Laryngol. u. Rhinol., 1920, 33:751-762.

Horn, H.: The etiology and treatment of ozena; a preliminary report. Jour. Am. Med. Assn., 1915, 65:788-793.

A contribution to the bacteriology of the so-called coccobacillus fetidus ozena (Perez), with additional notes on the treatment of clinical ozena by means of polyvalent vaccines made from the same organisms. Laryngoscope, 1917, 27:208-210.

Lautenschläger, A.: Operative Behandlung atrophischer Zustände des Naseninnern. Berl. klin. Wchnschr., 1917, 54: 445; 687.

Lorie, A. J., and Lux, P.: Atrophic rhinitis and ozena: Halle operation. Ann. Otol. Rhinol. and Laryngol., 1924, 33:1263-1270.

Perez, F.: Ozene et paraozene. Ann. d. mal. de l'oreille, du larynx (etc.), 1922, 41:571-578.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 12511

MEDICAL DEPARTMENT

The patient, an American clergyman, first entered the hospital in December, 1920, at the age of fifty-one.

The family history obtained at that time was unimportant. He had been married for sixteen years. He had no children. His wife had had three miscarriages.

The past history is unimportant save for a fracture of the right femur in 1915, followed by some slight permanent shortening of that leg, a sudden stroke of paralysis with involvement of the right side in 1917, and an attack of jaundice in 1919.

He entered the hospital at this time because of pain in the right lower quadrant with slight nausea and some fever, beginning four weeks before admission and lasting several days, recurring as chills and fever on several occasions, with pain eventually localizing in the left groin and radiating into the penis. He had frequency of micturition and incontinence of urine.

Physical examination showed a well developed and nourished man with a slight right facial palsy. Head otherwise negative. Neck negative. Apex impulse of the heart neither seen or felt. Sounds regular, not rapid, of good quality. The pulmonic second sound was greater than the aortic second, which was slightly accentuated. The artery walls were not thickened. Blood pressure 136/88. The lungs were clear. The abdomen was negative. The right leg was an inch shorter than the left. The right knee-jerk was greater than the left. There was no clonus or Babinski.

The urine showed from a large to a very slight trace of albumin and at the time of entry many white corpuscles. A Wassermann was negative.

For about five weeks he ran an irregular fever, with a leucocyte count averaging from 7000 to 17,000.

X-ray of the urinary tract showed no evidence of stone in the kidney or gall-bladder.

Cystoscopy showed redness around both ureteral orifices, somewhat more marked on the right. Lying free in the bladder was a minute stone the size of a pinhead. The diagnosis of pyelitis was made. For treatment fluids were forced. Urotropin was given. The pelves of the kidney were washed out several times with one per cent. silver nitrate solution. After a nine weeks' stay in the hospital he was discharged very much improved.

He next came to the hospital in December, 1924. The story was then that he had on the whole got on very well after leaving the hospital, with no urinary symptoms whatever and with fair general health, except for occasional days when he seemed to lack energy. He had purposely lowered his activity ever since his stroke of six years before and was accustomed to take a fifteen minute nap several times a day. A year and a half before this second entry he had noticed a marked increase in thirst and a polyuria, but no loss in weight. His local physician found sugar in the urine and put the patient on a low carbohydrate diet, which however has always been an approximation and never actually measured. The symptoms of polyuria and thirst disappeared under this régime, and the doctor told him that his urine had become sugar free.

He entered the hospital at this time (December 1924) because his doctor felt that his tonsils might be a source of trouble and that they should come out. His doctor had found that his blood pressure had occasionally been elevated, as high as 150 systolic. He had no symptoms referable to his tonsils, no sore throat, and no upper respiratory symptoms save a slight catarrh.

Physical examination at this time showed the same neurological findings as before. The heart was essentially as at the previous note. The blood pressure was 160/100.

The urine showed no albumin, no sugar, specific gravity 1.010 to 1.012, with nothing remarkable in the sediment. The red count was 3,700,000, hemoglobin 70 per cent. A Wassermann was negative. The non-protein nitrogen was 35 milligrams. After a carbohydrate meal a diabetic type of blood sugar curve was obtained.

A throat consultant reported on the tonsils as follows: "Pharynx and tonsillar pillars injected. Pus and miliary abscesses in the right tonsil. Some depression in the left tonsil. All I can say is that this type of tonsil in a man of fifty-five is not doing any good. Advisability of their removal should rest on your findings. We must consider his tonsils as a possible or even probable focus of infection."

After a study of a week, during which time he was kept sugar free on a diet as high as 70 of carbohydrate, 70 of protein and 180 of fat, it was concluded that although he undoubtedly had diabetes it was of a very mild grade. His blood sugar at entrance was 198, soon dropping to 132 and then to 114. After carefully weighing the

evidence it was decided not to take his tonsils out, because there was no positive proof that they were doing any damage, but instead to reobserve him some months later to discover whether his diabetes had progressed and with the idea of doing tonsillectomy then if necessary.

He entered the hospital a third time in July, 1925, for three days to have the diabetic situation checked up. He appeared to be able to stay sugar free on a diet of 55-65-150, on which he was discharged.

His physical examination was as before. His blood pressure was 150/90.

The urine showed the slightest possible trace of albumin, in the sediment a rare white cell. The red count was 4,900,000, hemoglobin 90 per cent. The non-protein nitrogen was 23 milligrams.

The patient did not again enter the Massachusetts General Hospital, but was seen by the writer on June 11, 1926, at his home upon the request of his local physician. He had been suffering for three or four days with a feeling of slight fatigue and with dyspnea which came in attacks not related to exertion. He had been put to bed by his local doctor. His complaints, aside from the dyspnea, were quite indefinite. He was not able to say himself what was bothering him, but simply that he did not feel quite right. His color was fairly good, and as he lay in bed there was no obvious respiratory embarrassment. His heart was beating regularly, rapidly, at the rate of 110, with sounds of rather poor quality. The left border of dullness was two centimeters outside the midclavicular line. No murmurs were heard. The lungs were clear. The belly was negative except for an obese wall. The extremities were negative except for the shortening of the right leg and increased knee-jerk on the right noted previously. There was no edema.

Digitalis was started and a total of twenty-one grains administered in the course of three days, after which he was given a daily ration of one grain and a half. He was kept in bed for two months, during which time there was a very slight improvement subjectively and essentially none in the physical examination. Neither the rest in bed nor the digitalis had any influence on his pulse rate, which remained usually in the neighborhood of 100. His blood pressure on several occasions was in the neighborhood of 160/100.

His urine remained free from sugar except directly after meals, when there would occasionally be a green test with Benedict's.

From August 10 on he was very slowly got out of bed and finally out doors for very short walks on a level. In the middle of August, since it was very desirable to get him away from the worries being close to his work entailed, it was decided to allow him to go to a relative in Chicago for a prolonged vacation. This he did, with apparent success. The journey both ways was made comfortably, a nurse going with him, and the vacation itself, during which time he remained on a semi-invalid basis, taking short walks and sitting on the veranda most of the day, was a pleasure to him and he felt that it had given him some added strength. He arrived back in Boston on his way to his home on October 9 and was seen by the writer. He reported that he had continued to take a grain and a half of digitalis daily and had adhered to his regular diet, as noted previously. He had dyspnea on slight effort or on excitement, but no cough or sputum, no pain in the chest, no edema. There were no gastro-intestinal symptoms and no urinary symptoms. His color was fairly good. The pulse was rapid, rate 110, regular. The left border of dullness was two centimeters outside the midclavicular line. The heart sounds were regular but of poor quality. When he talked he was apt to get excited and then got decidedly breathless.

On October 13, while stopping at a hotel in Boston, he telephoned the writer at 12:30 a.m. and said that he felt restless and that he could not sleep, but in other respects felt all right and had no shortness of breath, pain in the heart, or cough. He said he would like to have some medicine to make him sleep. Five minutes later the operator at the hotel reported that the patient's wife thought he had died. The writer arrived fifteen minutes later to find the patient living but evidently *in extremis*. He had gone to the toilet directly after telephoning and while there had been seized with a severe pain in the region of the heart. He had been helped back to bed and then collapsed. At the time he was seen he was deeply cyanosed, breathing stertorously, pulseless at the wrist, with heart sounds that were hardly audible. His color rapidly gave way to pallor, his breathing became very halting, and five minutes later he died.

Discussion

By James H. Means, M.D.

Notes on the History

I have two patients' stories that I wish to take up today, because they have some interesting points of similarity and some of dissimilarity. The thing I am chiefly interested in is the cardiac problem in each of them. I will ask Dr. Paul D. White to discuss them both, and as each has a long past illness I will go through the histories rather rapidly, so that we can get to the important points without too much delay.

The story of the illness that brought this first patient to the hospital is suggestive of some acute infectious process in the urinary tract. The points of interest back of that are, first of

all, that he had had a hemiplegia, and we think of the possible causes of hemiplegia in a man of middle age. Syphilis would come into our mind. We note the negative Wassermann and also note that his wife had three miscarriages.

After having had a pyelitis in 1920 he apparently developed diabetes in 1924.

A systolic blood pressure of 150 is certainly not high for a man of this age. In 1924 the blood pressure is increased over the level recorded in the hospital in 1920.

At this time we have a second negative Wassermann.

A diabetic type of blood sugar curve means that it stayed up more than two hours and I suppose reached a level of more than 180 or so.

We all had the feeling that it might be meddlesome to do a tonsillectomy on this man. His tonsils were giving him no symptoms. He was arteriosclerotic and he had this diabetes. He had had a pyelitis. Altogether it impressed us that he was the type of man we were anxious not to do any unnecessary surgery upon, even a tonsillectomy.

The red count was a gain over last time.

This brings us up to the illness I wished chiefly to present here. The impression I had of him up to this point was that he was a man looking older than his years, who was undoubtedly arteriosclerotic. He had had a shock in 1919 which I did not think was syphilis. There was no source in his heart for an embolus to come from, and it seemed to me that in all probability he had had a slight cerebral hemorrhage.

The jaundice in 1919, about which I know very little, is worthy of noting.

The pyelitis was an infection which at least did not do him any good, and may have done him harm.

The development of diabetes in an arteriosclerotic person who has already had a shock, who has a mild grade of hypertension, seems to be part of the picture of general arteriosclerosis. We sized up his diabetes as being relatively mild. He had a relatively low threshold and a low blood sugar. Dr. Brigham agreed that his diabetes was of what we call the arteriosclerotic variety. He never required insulin, and it really was never a very important part of the picture.

The condition which kept him in bed from June 11 until August was not entirely easy to size up. He could not tell us very much about his exact symptoms, but he knew he did not feel right. He had these attacks of dyspnea which were not very severe. He had a rapid pulse which as I have pointed out usually showed no tendency to come down with prolonged rest or with digitalis; and that was substantially all. He was obviously ill, and I felt that he must have some myocardial weakness. But he did not have congestive failure in any sense. He never had any edema that I could discover. He had no precordial pain, no precordial tenderness, nothing that suggested an attack of angina pectoris or any anginoid symptoms of any kind up to that time.

The question therefore came up as to therapy, and it seemed more or less inhuman to keep him in bed for the rest of his life. He wanted to get up. I did not know precisely what the condition of his heart might be. He was down on the Cape and I could not get cardiograms and things of that sort, and the local doctor and I thought it was proper to get him up slowly and see what would happen. We did so, and he seemed to be no worse than before. His pulse stayed at about the same level. He could not go upstairs without getting dyspneic, but could get about on a level. It was very desirable to get him away from his parish. He had an opportunity to go out to Chicago and live a life of comfortable invalidism out there with some relatives, and we thought it was proper to let him do so.

DIFFERENTIAL DIAGNOSIS

The thing that impressed me was the multiple evidence of sclerosis in this man. Added to the ones already mentioned we have now possible evidence of an arteriosclerotic process in his heart. The pulse not responding to digitalis or rest made me feel that he had a myocardial disturbance of a grave sort, and the final event of a sudden pain in the heart suggested very strongly that he had had an occlusion in his coronary arteries. I did not get a blood pressure the last time I saw him. It would be of a good deal of interest to know what his blood pressure was directly before this attack.

I made the following diagnosis on his death report: Arteriosclerotic heart disease, hypertrophy and dilatation of the heart, coronary thrombosis, diabetes, hemiplegia, right, slight, probably from an old hemorrhage.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Coronary thrombosis.

DR. JAMES H. MEANS' DIAGNOSIS

Arteriosclerotic heart disease.
Hypertrophy and dilatation of the heart.
Coronary thrombosis.
Diabetes mellitus.
Hemiplegia, right, slight, due to old cerebral hemorrhage.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Arteriosclerosis of the coronary arteries and the aorta.

2. *Secondary or terminal lesions*

Chronic fibrous myocarditis.
Infarct of the heart (old).
Chronic passive congestion of the viscera.

3. *Historical landmarks*

Chronic cholecystitis and cholelithiasis.
Chronic vascular nephritis (slight degree).
Chronic focal non-tuberculous inflammation of the lungs.

DR. MALLORY: The heart in this case showed a moderate enlargement, both ventricles being somewhat dilated. On the anterior surface of the left ventricle, two and a half millimeters from the apex and one from the median septum, was a soft white area covered with fibrous tissue. The right ventricle wall measured three millimeters, the left averaged fifteen, but at the point of the scar referred to the total width was only half a centimeter, and the muscle was entirely replaced by white fibrous tissue. Elsewhere the myocardium was normal in appearance. The valves were normal.

The first centimeter of the left coronary artery showed very marked sclerosis with thickening and an atheromatous plaque in the intima, a slight degree of calcification. Slightly beyond this point there was another area in which the lumen of the coronary was decreased to about one-fourth its average diameter. It was however still patent, and we found no thrombi. The right coronary artery was free from involvement for the first three centimeters, but at that point there was another small patch of sclerosis and an approximately equal degree of narrowing of the lumen.

The aorta showed very marked sclerosis in all portions including the ascending, with well marked atheromatous plaques, calcification, some fairly deep ulcerations, and some thrombi adherent to the ulcers.

The liver was rather large and congested. The gall-bladder was very small and shrunken, measuring two by one by one centimeters. The walls were thickened to a width of about four millimeters, the cavity almost obliterated. One small stone one millimeter in diameter was found. The bile-ducts were patent throughout. There were also very marked old chronic adhesions between the gall-bladder and the duodenum and omentum.

The lungs showed two very peculiar lesions. On the diaphragmatic surface of each lung there was a hard, irregular infiltration extending in towards the root of the lung for a distance of about three centimeters. There was no bronchiectasis, and it did not look in the least tuberculous.

I think, so far as we can draw conclusions from the necropsy, his type of jaundice is explained by the findings in the gall-bladder, and the fibrotic area in the heart must represent an old infarct which is of some years' standing.

DR. MEANS: What do you think was the immediate cause of death?

DR. MALLORY: That is rather hard to say. There was no demonstrable cause at the necropsy. Both coronary arteries were patent, and there was no thrombosis and no embolus. On the other hand, each coronary was at at least one point very markedly diminished in diameter, and if one wants to imagine a spasm, it could very easily have taken a very slight degree of that completely to obliterate the lumen of the coronary for the matter of a few minutes. I do not think that the time involved between his seizure and his death—it was less than thirty minutes, wasn't it?

DR. MEANS: Yes. The whole length of time was not much more than twenty minutes or twenty-five at the outside.

DR. MALLORY: We should have to have a period of several hours in order to get any definite appearance in the myocardium post-mortem that would allow us to decide. No necrosis had occurred there before the general death of all the tissues.

DR. CABOT: This thin patch was obviously old—you cannot say it was months or years, but it was not weeks anyway?

DR. MALLORY: No.

A PHYSICIAN: Do you think that thrombi could be missed in a case like this—a thrombus easily dislodged or overlooked?

DR. MALLORY: That is quite possible, especially under unfavorable conditions. This was done outside the hospital.

A PHYSICIAN: That has been suggested in the literature, that such an occurrence might explain some of the absent findings. But that is only a theory.

DR. MALLORY: That is quite possible. I should be the last to question it.

A PHYSICIAN: May he not have had a cerebral embolus at the time of the hemiplegia?

DR. MALLORY: I think that is quite possible. He had sufficient arteriosclerosis to make that possible. None of the thrombi in the aorta was large, but there were small ones in the ascending aorta. It is quite possible that one of them might have shot up.

DR. CABOT: He never had any arrhythmia?

DR. MEANS: Not that I know of.

DR. CABOT: I should like to ask Dr. White whether this steady tachycardia that he had had been noted before in connection with any of these myocardial scars?

DR. PAUL D. WHITE: I think it has, but whether by careful study I don't know. I believe it would not do any harm to investigate the relationship. In my experience it has not been unusual to find a tachycardia unexplained

except by myocardial involvement, sometimes with poor sounds or with gallop rhythm. Usually we cannot get the rate below 100 by any procedure until the myocardial condition improves.

DR. CABOT: Can you see any way in which the lesions here found ought to produce tachycardia?

DR. WHITE: The tachycardia may be due rather to the general coronary narrowing than to the lesion itself,—poor myocardial blood supply. The point comes up as to whether or not death may have resulted from an abnormal rhythm. It seems to me unlikely, because the death was gradual—ventricular fibrillation for example would cause immediate death.

DR. MEANS: He was almost pulseless and his heart sounds almost inaudible when I got there.

DR. WHITE: But you could hear them.

DR. MEANS: Just barely. I did not notice any arrhythmia.

DR. WHITE: Was the rate rapid?

DR. MEANS: Moderately.

DR. WHITE: Very rarely ventricular fibrillation has been found electrocardiographically, but in the few patients whom we have electrographed at the time of death we have not found it. There has been rather a depression of the sino-auricular and A-V nodes with block and gradually a cessation of first auricular and then ventricular contraction or vice versa without ventricular fibrillation. But there has been a feeling that a possible cause of sudden death in heart disease may be ventricular fibrillation. It does not seem as if the terminal tachycardia in this case could have been responsible for death, because hearts may sustain very rapid rates—ventricular rates of 270 or 300 per minute have been sustained with complete recovery.

DR. MEANS: He had pain, his wife said, then toppled over.

DR. WHITE: Angina pectoris would be the best explanation of his sudden death, I suppose, whatever angina pectoris is.

DR. CABOT: This is the type of case that you classify as arteriosclerotic heart disease, isn't it?

DR. WHITE: Yes. There has been a systolic blood pressure noted of 160 millimeters, which may mean some hypertension, but arteriosclerosis is mostly responsible for this man's heart disease.

DR. MEANS: 160 was the highest; it was often-times lower.

Just one small point: it was interesting that we have here a combination of gall-bladder disease and diabetes. We have already talked about the possible connection between the arteriosclerosis and the diabetes.

CASE 12512

MEDICAL DEPARTMENT

An American business man was first seen in December 1922, at which time he was sixty-one years old.

His family history was unimportant.

His wife was living and well. She had had one miscarriage and lost one child at birth. There were two adopted children.

He could recall no illnesses except smallpox in childhood. He denied venereal disease. The trouble for which he sought relief had begun two years previously. At that time he had been working very hard at his business and under considerable outside strain because of a great many charities and war activities in which he was engaged. The first symptom was the feeling at night of what he described as a hot liquid rolling back and forth across the upper abdomen. There was no pain, but there was occasional belching of gas. There was no nausea or vomiting. The gurgling sensation at first did not occur regularly, but eight months before admission it became worse and he began to lose his appetite. In July, six months before admission, his local doctor told him he had a peptic ulcer and put him on a diet of milk, crackers, cereal and broth. On this he improved somewhat, and after a vacation in the autumn of 1922 he had occasionally gone on a general diet without ill effect. He consulted the writer not because his symptoms had returned, but because he wanted to have his condition studied. While on the limited diet his only symptoms were rare belching and some gas in the bowels, with the passage of considerable flatus. There was no nausea or vomiting, but an occasional eructation of acid gastric juice. His bowels were regular and his appetite good. He had lost no weight. His usual weight was 148 pounds, which was also his present weight. He never had had black or bloody stools or been jaundiced. He said that he had never, during a long business career, been obliged to stay away from his office on account of his health. There were no symptoms referable to the cardiorespiratory, genitourinary or neuromuscular systems. He slept well and took occasional exercise in the form of golf.

Habits. He smoked ten or twelve pipes or cigars a day, no cigarettes. He took alcohol very rarely.

Physical examination showed a well developed and nourished, rather short, stocky man with rather high color. The right pupil was very slightly irregular; both reacted normally. The tongue was coated. The throat was not remarkable. Several teeth were missing; a good many gold caps. Breath somewhat offensive. Neck negative. Lymph nodes not enlarged. Skin clear. Heart, lungs, abdomen and extrem-

ities entirely negative. Arteries not noticeably thickened. Blood pressure 128/90. Rectal examination showed some very small external hemorrhoidal tabs. Digital examination negative.

The urine showed a specific gravity of 1.020, no sugar or albumin. A large amount of white amorphous sediment. No cells or casts.

X-ray of the gastro-intestinal tract by Dr. A. W. George: "No evidence of ulcer at the present time, at least gross ulcer."

The writer expressed the opinion at this time that the findings did not warrant the positive diagnosis of peptic ulcer, although he admitted that it might be present. He recommended an increase in the diet and urged that the patient cut down all activities except those strictly confined to his business and take somewhat more regular exercise. It was also recommended that he see his dentist again, have his teeth X-rayed, that any infection that might be discovered in or about his teeth be cleared up, and that he be given the necessary bridge work to give him adequate chewing apparatus.

September 6, 1923, a friend of the patient reported to the writer that the patient had been getting on very well and was doing his work right along.

He was next seen by the writer January 5, 1926. The story was that in January and February, 1923, he had gone to California for a vacation and then had come home and gone to work. He had followed the diet recommended and got along free from all symptoms until January 1925. At that time he began to lose weight and to have hard lumpy stools. May 30, 1925, he went to Battle Creek and stayed there until July 23, first for examination and then for treatment. The result of the examination there as reported to me was that his stomach did not empty for eight hours and that he had the scar of an old ulcer in his duodenum. While out there about twelve teeth were extracted by a dentist not connected with the Sanitarium, and false teeth were supplied. The treatment at the Sanitarium consisted of diet, baths and colonic douchings. Petrolatum was given, and he got so that he regularly passed a formed but very soft stool. The outline of the findings at Battle Creek report that two examinations of the stomach showed in the fasting contents a trace of blood and some mucus, and in the test meal free hydrochloric acid 18, 10, 50, 52, 46, 18, the total acid 34, 16, 62, 68, 66, 38. The urine was not remarkable. The feces were formed and brown, showing no mucus. Guaiac was positive on one occasion. The stools were said to show a bad type of intestinal flora. The blood showed 4,800,000 reds, 85 per cent. hemoglobin. A Wassermann was negative. The non-protein nitrogen was 37 milligrams. X-ray examination of the stomach showed peristaltic waves normal, passing out without hindrance on both curvatures to the pylorus. There was a constant deformity of the duodenal bulb characteristic of ulcer. The emptying time of the stomach at this examination was about six hours.

Since leaving Battle Creek he said that he had been back at his office and two-thirds of the time felt perfectly well. When not well he had attacks in which he had a feeling of distension in the upper abdomen lasting from a half hour to an hour. If he lay down it passed off. At night from one to three a.m. he often had a similar sensation. Since he had petrolatum at Battle Creek his bowels had been regular. He had no nausea, vomiting or jaundice. His weight went up to 140 pounds while at Battle Creek; now 130. His appetite remained good. Three weeks ago he began to have more continuous trouble. He had dull pain in the epigastrium, apparently related to meals, coming two to four hours after meals. Lying down helped this pain. He was still free from nausea and vomiting. The bowels were regular. There was some flatus, but no belching. The abdomen was never distended. He was on a soft solid diet with frequent feedings, and took a mixture of bismuth and soda p.r.n. for distress, with some temporary relief.

Physical examination showed much the same general appearance as before. The tongue was coated. The heart and lungs were negative. The abdomen was slightly pendulous, soft, no masses, tenderness or viscera felt, no gas. Rectal examination was entirely negative. Rectum empty.

X-ray examination by Dr. Holmes was made on January 11. There was no delay in the esophagus. Nothing unusual in the chest. The stomach was in the usual position and filled normally. There was no irregularity of the gastric outline or peristalsis, and no gastric stasis. The pyloric sphincter and first portion of the duodenum however presented a constant and fairly typical irregularity. The head of the barium column reached the middle of the transverse colon in six hours and the rectum in twenty-four hours. The cecum and colon were not remarkable. The appendix was seen fairly well filled, movable and not tender. "The findings are those of a duodenal ulcer without gastric stasis."

In view of the two positive X-ray examinations it seemed impossible to avoid the diagnosis of peptic ulcer. It was decided that the treatment should continue to be medical, because there was no evidence that the ulcer was producing stasis or that it was bleeding, because his symptoms were removable by dietetics and because the location of the ulcer in the duodenum with a perfectly normal stomach made the possibility of cancer very remote. He therefore was to continue on his five-meal bland diet,

taking alkali powders after each meal. He was also urged again to cut down his work if possible.

October 8, 1926. The patient reported that since the time the last note was made he had remained on the diet prescribed and that he had been very comfortable until July 1926. During the month of June his diet had included chicken, a few vegetables and purées, and he had felt better than for a long time. In July, however, he committed the indiscretion of taking some lobster salad followed by some strawberries, which produced some definite epigastric distress lasting not more than a couple of days. A few days after this episode he took a clam chowder, which upset him a great deal. He had dizziness, nausea, diarrhea and a little fever, and was miserable for about two weeks. He had never felt quite well since this. He had been fairly comfortable, but had not had such good nights, being kept awake by gas in his stomach and epigastric discomfort. September 23 he went to New Hampshire for his vacation. The first four days he felt remarkably well and played as much as eighteen holes of golf. Another chowder however started his epigastric distress, which continued for several days. On October 3 he returned from his vacation. The pain in his epigastrium tended to increase and he vomited. He was also constipated, and took some castor oil which worked twelve hours later. On the 5th he vomited again. He went to bed and stayed there until to-day, when he vomited some pinkish-brown material, and thought it best to come into the hospital for study.

Physical examination showed the chest and abdomen entirely negative. There was no tenderness at all in the epigastrium. No masses could be felt.

The urine was pale, cloudy, alkaline, specific gravity 1.013, the slightest possible trace of albumin, rare hyaline casts, a few leucocytes, amorphous phosphates. The blood showed 4,300,000 reds, hemoglobin 75 per cent.

The patient was put to bed on a régime of hourly feedings of milk and cream mixture three ounces alternating with warm gruel flavored with milk or cream six ounces. October 9 he had a very poor night, vomiting several times. The vomitus consisted of the milk and cream mixture and gruel, was very slightly pinkish, showing a faintly positive guaiac test. He had had no pain, merely gas and nausea. The temperature was normal, the pulse 62. The feedings were stopped for six hours, then an ounce and a half of milk mixed with an ounce and a half of White Rock every hour was begun. A surgical consultant believed that no surgical emergency existed and that after his gastric digestion had been straightened out the matter of posterior gastro-enterostomy might be considered. October 10 he again had a poor night, vomiting several times. The vomitus this time was frankly blood streaked, but not uniformly pinkish. An enema gave a black liquid stool showing a negative guaiac. The belly was soft; no tenderness, no masses. Everything by mouth was stopped. He was given cracked ice only. He was given one-sixth of a grain of morphia and 100 cubic centimeters of three per cent. glucose solution by rectum every two hours. October 11 he had another poor night, much retching but no actual vomiting. He was seen by a second surgical consultant who agreed with the probable diagnosis of peptic ulcer and believed that no surgical emergency existed, but that it seemed very likely he would require a gastro-enterostomy soon, and that since it was possible that he would go on vomiting for some time, thereby getting into a considerably worse condition, it would be desirable, if the patient desired operation at all, to proceed at once.

October 12 laparotomy was done. It disclosed an annular lesion in the neighborhood of the pylorus, hard and nodular, extending half and inch along the lesser curvature of the stomach and about three-quarters of an inch into the duodenum. The surgeon thought it felt very much like cancer. He resected about a third of the stomach and anastomosed the lower end to the cut end of the duodenum.

October 13. 9 a.m.: Making a good operative recovery. 1:30 p.m.: Patient suddenly developed an irregular pulse. 2 p.m.: Seen by the writer. Heart obviously fibrillating, with a rapid rate and marked pulse deficit. The heart was not enlarged. The sounds were of poor quality. His condition was such as to cause considerable alarm. There was no dyspnea. The lungs were clear. Digitalis was started at once, digitalin being given subcutaneously. 8 p.m.: Heart now regular, but very rapid (140), almost imperceptible at the wrist, with sounds of poor quality. His condition seemed very critical. He had now had six grains of digitalis.

October 14. 7:30 a.m.: Condition perhaps a shade better, but still alarming. Pulse very rapid and thready at the wrist. Color good. Breathing through the night had often been Cheyne-Stokes. 9 a.m.: Seen by Dr. White. Electrocardiogram taken. This showed sino-auricular tachycardia. Dr. White believed this was of extracardiac origin. He expected the digitalis to accomplish very little, but thought it ought to be continued. 12:30 p.m.: Transfusion of 500 cubic centimeters of blood was done. After it his color improved and his pulse volume increased perceptibly. Digitalis was continued; with the dose given at two o'clock a total of twenty-one grains had been administered.

October 15. 9 a. m.: Condition not so favorable. Pulse still high. Heart action poor,

sounds regular, no friction rub. Breathing fairly good. Color fairly good. No edema. 7 p.m.: Respiratory rate going up. Almost pulseless.

October 16 the condition grew steadily worse and at one a.m. he died. At no time was there any rattling in his chest. The abdomen throughout was entirely negative. The highest temperature at any time before or after operation was 101.2°, on one occasion October 14. The rest of the time the temperature had been within normal limits.

Discussion

by James H. Means, M.D.

Notes on the History

The fact that he has never had to stay away from his office on account of his health is perhaps of a good deal of importance.

It seemed to me that this story was that of a tired business man who had been carrying a good deal of responsibility and who had gastro-intestinal symptoms of a very definite nature, and that, coupled with a negative X-ray, made it impossible for me to agree with his doctor that he had an ulcer. He was in a big financial house and was carrying all sorts of extra loads and responsibilities, and I suggested that if he would cut these out it would be a fair compromise. But he did not do it.

In January 1925 his symptoms still did not sound particularly like peptic ulcer.

The trace of blood in the fasting contents is perhaps of some interest.

I do not know much about "intestinal flora," but at Battle Creek they examine the relation between the number of Gram-negative and Gram-positive organisms and draw some conclusions from that.

He began to have more continuous trouble in 1925 or January 1926. This is the first time that it has been related to meals.

Dr. Holmes: This X-ray report was made on the fluoroscope examination and not on the films. All through this X-ray examination one thing should be noted. He has considerable deformity of the duodenum without stasis and without increase of gastric peristalsis. One would think that with that deformity and with ulcer he would have either stasis or increased peristalsis. It would make us think there might be something wrong in the interpretation. It might be malignancy or it might be spasm. I cannot add anything from the plates. We would never quite feel justified in making a diagnosis of cancer of the duodenum, because it is so extremely rare. So that whenever we get a constant deformity of the duodenum we are inclined to call it ulcer. It is a fact, however, that he should either have hyperperistalsis or stasis.

Dr. Cabot: Is there an X-ray recognition of a healed ulcer?

Dr. Holmes: No.

Dr. Cabot: You don't make that diagnosis?

Dr. Holmes: No.

Dr. Means: Of course I finally thought he had a peptic ulcer. I did not think there was any mystery about the diagnosis at all, but that I had made a mistake when I first saw him in thinking that he did not. And I thought he had an acute flare-up with a vicious circle with vomiting etc. as these patients often do. It did not occur to me that he had stasis. He was not bleeding profusely, but this vomiting of brownish-pink material probably indicated that he had an ulcerated lesion somewhere. The black stool was due to bismuth.

That was the situation just before operation. Everybody thought it was ulcer,—the two surgical consultants and myself. I told the family that it was an ulcer. Asked if he had cancer I said it was possible, but I thought a very remote possibility.

Operation

The usual upper abdominal incision. There was a large growth in the pyloric end of the stomach in the region of the pyloric ring, almost completely obstructing the pylorus. We were unable to determine which side of the pylorus it was on. This growth was removed by the so-called Shoemaker operation, partial gastrectomy, in which an end-to-end anastomosis of stomach and duodenum was done. The growth was very adherent to the pancreas. There were several glands along the lesser curvature. It was very difficult to determine by gross examination whether the growth was ulcer or malignant; it seemed probable that it was malignant, because it was so hard.

Further Discussion

Dr. Mallory: Unfortunately I have not the record of the specimen here, but as I remember, it showed an infiltrating annular mass running from just inside the pylorus about as described in the surgical record, perhaps the last centimeter of the stomach, the pylorus and the first centimeter or two of the duodenum involved. The pylorus was still open. We could pass a finger through it, although there was a slight degree of contraction. We made a frozen section at the time and found infiltrating cords of epithelial cells running through rather scirrhous stroma, and made a diagnosis of carcinoma at that time.

Dr. Means: Would it be fair to say that this was carcinoma of the duodenum?

Dr. Mallory: I do not think we can be sure of it in this case. The lesion certainly extended a little beyond the pylorus in each direction, so

it may have started in the stomach and grown into the duodenum. Where the primary focus was I cannot say with certainty. There is one other point one could argue from however: that the surgeon found the growth extremely adherent to the pancreas. That was not an infiltration of cancer into the pancreas. We found no cancer in the pancreas at all. The adhesions to the pancreas were old inflammatory ones. So that it is fair to argue from that, that perhaps the cancer developed from a previous ulcer. I do not think we have any positive proof of it, but that would fit the picture a little better perhaps than anything else.

DR. MEANS: The lesson to be drawn is that a lesion may be situated, so far as X-ray examination goes, in the duodenum, and the symptomatology may be of long duration and resemble ulcer as this did finally, although I do not think it did in the beginning, and yet prove to be cancer.

The heart began to fibrillate apparently at two o'clock on the day following operation, and by eight resumed a regular rhythm. The man had been doing very well after a severe operation on his stomach, and then suddenly developed an attack of fibrillation which lasted several hours, then stopped, and was replaced by a very rapid and poor quality pulse, so that something very acute had happened. This was a man, I might point out, who had never had anything indicating disease of his cardiovascular apparatus in the past of any kind or description, so far as I know,—no etiology. One pupil was irregular, but no symptoms of syphilis had ever been found and the Wassermann was negative.

Transfusion was done with the thought that possibly this state that he was in was in some way an unusual type of shock, and that something pretty drastic had to be done in a hurry, and that seemed as reasonable as anything else. It was not done for hemorrhage. There was no evidence of his having lost any large amount of blood. His color had not been good, but it was not the extreme pallor of massive hemorrhage. He made a brief rally after this transfusion, but it was very brief.

DIFFERENTIAL DIAGNOSIS

This is all the data I have. He did not respond to digitalis and he only temporarily responded to transfusion. So far as we could make out he had no infection. No white count was done for the reason that it did not seem to be indicated. I supposed that something had gone wrong in his heart in all probability, that the cause of this death was intracardiac, and that possibly here as in the first case might be also coronary thrombosis. But we really had no clear idea of what he did die of.

Do you care to make a diagnosis, Dr. Cabot, on the cause of death?

DR. CABOT: I think I should bet that nothing particular would be found. It is the kind of death that often happens after an operation for cancer without our knowing why.

A PHYSICIAN: Could he have a coronary thrombosis without pain?

DR. WHITE: Such cases are on record. Coronary occlusion probably is not very rare, but if it occurs suddenly pain almost invariably results. There are rare cases of sudden occlusion and occasional cases of very gradual occlusion in which there is no pain. This is a very important reason why the present case should not have been considered one of coronary occlusion. There is one quite remarkable case reported by Gross, with complete occlusion of the right coronary artery in an elderly woman who never had any symptoms or signs of heart trouble. She died of something else—pneumonia I believe—and at the post-mortem examination her heart was all right except for complete occlusion of the right coronary artery which had come on so slowly that her myocardial circulation was completely taken care of.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the stomach.
Myocardial insufficiency.
Probably acute coronary thrombosis.
Operation, resection of stomach.

DR. JAMES H. MEANS' DIAGNOSIS

Carcinoma of the stomach.
Acute coronary thrombosis.
Cardiac failure.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Carcinoma of the stomach with metastasis to a regional lymph node.

2. *Secondary or terminal lesions*

General peritonitis.
Fat necrosis.
Acute focal myocarditis.
Bronchopneumonia.

3. *Historical landmarks*

Diverticulitis of the bladder.
Adenomatous hypertrophy of the prostate.

DR. MALLORY: On opening the peritoneal cavity in this case a considerable amount of yellow-brown turbid fluid escaped from a well localized cavity in the upper portion of the abdomen bounded by the dome of the diaphragm, the superior surface of the liver, the stomach, duodenum and omentum. The latter was firmly adherent to the wound and shut off the cavity

from the greater peritoneal cavity. Cutting through showed the lesser cavity to be also free from fluid. In the walls of the cavity which contained the fluid there were numerous yellowish-white small opaque areas in the fatty tissue, typical fat necroses.

The stomach showed the sutures of the recent operation, which in general were firm. There was one long line of sutures along the anterior surface of the stomach perpendicular to a ring of sutures which encircled the amputated portion of the stomach and sewed together the anastomosis with the end of the duodenum. The sutures of this ring were imbedded in tissue which was very friable and partially necrotic, pulling apart with very slight tension. There was no very large or coarse leak, but at the upper end of the line was a small abscess cavity, and a fistulous track extended down posteriorly to the duodenum under the head of the pancreas. This was lined with necrotic tissue, and numerous areas of fat necrosis were evident along the track.

The surface of the pancreas showed a few sutures near the head, and there were some fat necroses about them. Several enlarged lymph nodes were found about the head of the pancreas, appearing in gross like acute inflammation, without evidence of metastases from the cancer.

The lungs showed large quantities of serous fluid which dripped from the cut surfaces, and in the upper portion of the left lower lobe was a patch of deeply congested firm tissue, obviously a fresh bronchopneumonia. There was a very small patch one centimeter in diameter at the right base also.

The heart weighed 300 grams, and was negative except for very slight thickening of the mitral cusps. The coronary arteries showed a slight degree of sclerosis, an atheromatous thickening of the intima for the first three centimeters on the left side, but there was very slight diminution in the lumen of the artery and no thrombosis. The aorta showed fairly marked sclerosis from the descending portion of the arch downward into the abdominal aorta.

The liver was not congested. The kidneys were very slightly granular, and there was congenital anomaly of the bladder, a large diverticulum about the orifice of each ureter.

Microscopic examination brought out two things that we did not make out in gross. In one of the lymph nodes from the head of the pancreas there was a very small, early, but I think definite, metastasis from the carcinoma. And the heart showed one small area not more than one-half millimeter in diameter of fresh necrosis. The muscle cells stained very poorly, vacuolated. The nuclei had largely disappeared. Some of the muscle cells were almost completely disintegrated. There was considerable hemorrhage about the necrotic cells, and beginning invasion with phagocytes. We found only that single area of heart necrosis. Its origin I am not quite sure of from the necropsy. It does not look like a vascular affair. I think it is quite possible that it was an acute toxemic thing from the process in the pancreas. If I remember right, Dr. Jobling in some of his work on experimental pancreatitis found evidence of general toxemia and sometimes necroses widely scattered in various tissues about the body. And that is the only suggestion I have as to the origin of this. Of course it is quite possible that where the lesion is microscopic in size there may have been many more which we did not find, and one of these may have involved the conducting tissues.

DR. MEANS: The diagnosis we made on the terminal event was wrong like the one made before operation. This man apparently died of peritonitis and with a symptomatology which I certainly would never have associated with peritonitis, because it was that, as I have said, of sudden fibrillation, poor heart action, fever only once, and nothing that pointed toward the abdomen at all. The abdomen was perfectly flat and non-tender, so it seems rather a striking *dénouement*.

DR. PAUL D. WHITE: This is a case to remember, I think, for a number of reasons. It was extraordinarily difficult as we have seen. I started out, when I first saw the patient, with the firm conviction that his heart was all right except that it was temporarily disturbed, and that something else was wrong; as Dr. Cabot has said, a serious surgical operation like this may cause trouble which is rather hard to define. The condition seemed to be allied to shock, possibly toxic, but I did not feel at first that it was the result of direct involvement of the heart. Therefore although I thought that it would not do any harm to continue the digitalis in case we were overlooking thrombosis, I believed that transfusion would help more than cardiac stimulation. And it did for a short time. Then the evening before death, when I saw the patient in consultation again with the surgeons and Dr. Means, and the surgical opinion was so strong that there could not be any surgical complication, since there was no hemorrhage, no evidence of peritonitis, and no ordinary surgical shock, I was confronted with the possibility of coronary thrombosis. There was some basis for this idea,—the earlier disturbance of rhythm, a rapid weak pulse, low blood pressure, and poor heart sounds in a man who was dying with no other apparent cause.

Of course the abdominal condition was very hard to diagnose, chiefly because the lesion was directly under the wound. Then too there were the dressings over the operation wound. It was a very localized affair. There was no general

peritonitis. I finally felt that the odds were even that coronary thrombosis was present as the cause of death. But now with this condition found at post-mortem examination the cardiac action is quite easily explained. It is the toxic effect of the abdominal complication on the heart's action. It seems to me it needs no further explanation.

I have not seen such a microscopic lesion as Dr. Mallory has reported, but I think that our custom here in the past of not making very complete microscopic examinations may have caused us to miss some such lesions before this. Perhaps they would be found only accidentally anyway. I do not see why such a lesion should come from coronary disease. The heart seemed otherwise on microscopic examination to be practically normal. Whether such a lesion could account for the paroxysmal auricular fibrillation is doubtful. Auricular fibrillation is not a rare complication after operation or other strain in an elderly person, and so it is not necessarily an indication of serious heart disease. The presence of such an arrhythmia therefore would not help us one way or the other.

It was our inability to make any diagnosis with rather prominent cardiac signs that led to the diagnosis of possible coronary thrombosis, even in the absence of pain. But it certainly was not a typical case clinically of coronary thrombosis. We should therefore remember that even though we are told by a surgeon that there must be some other cause than a surgical condition for such a cardiac state as this, we can point to this case at least as one example in which there was no evidence of a surgical complication which was actually present to disturb the circulation secondarily.

DR. CABOT: We have had a number of cases here at the post-mortem table in which there has been general peritonitis, not local peritonitis, a perfectly soft abdomen, and no symptoms of any kind. We are always forgetting those cases in people who have just been through an operation. We get the regular picture of general peritonitis as we get it in a perforated appendix or some such thing, and forget that we can have it with no symptoms at all.

I should like to ask Dr. Holmes if he has anything to say in retrospect?

DR. HOLMES: I can add but little to what I said a moment ago. There are two findings to which we should have given more weight,—the absence of hyperperistalsis and the absence of stasis, both of which should be present in a large duodenal ulcer. Not all duodenal ulcers have stasis, but practically all have hyperperistalsis. The other men who examined this case, both very good roentgenologists, did just what I did; they found the deformity which they placed in the duodenum, and on the basis of that they did not care to say that it was anything more than ulcer. I have never made the diagnosis of cancer of the duodenum. Here is a case where it has occurred. We had another case not long ago in this hospital, I believe, in which the diagnosis of cancer of the duodenum was finally made, but we were not able to make it by X-ray. Perhaps we have a distinguishing point here in that when we have a deformity of the duodenum which looks like ulcer and there is absence of hyperperistalsis and stasis we ought to consider cancer.

DR. CABOT: I understood Dr. Mallory to say that most of this was in the stomach.

DR. MALLORY: No, I did not mean to give that impression. There was a very small portion in the stomach, most in the duodenum.

DR. HOLMES: As I understand, the surgeon goes by the pyloric vein. We go by the contractive ring which forms its sphincter, and of course that would include quite a space on each side which would seem to be in the ring. I think it is perfectly possible that the mass interpreted as being in the duodenum was in the ring when you came to examine it anatomically.

CASE 12513

A CASE OF SPONTANEOUS FRACTURE OF THE HIP

ORTHOPEDIC DEPARTMENT

An Irish freight handler forty-three years old entered November 24 for injury of the left hip.

November 23 while at work and without any special injury or strain he felt something snap in his left hip. He fell to the floor, felt extreme pain in the hip and had been unable to use the leg since. He was brought immediately to the Emergency Ward and admitted to the hospital.

His family history is not important.

As a small child he was sickly. After this he was perfectly well until eight years before admission. At that time he was ill three weeks with pneumonia, and following this was weak and ill for several months. During this time he noticed that his legs began to bow and occasionally to be painful. During the next few years his legs had been increasingly painful. For several months he had had much pain at night, especially in the left leg. He thought that he had to have a larger hat now than formerly. His general health had failed markedly during the past few years. He denied venereal disease.

Examination showed a poorly developed and nourished man. The head was large, with prominent temporal bosses. The chest showed marked Harrison's groove. The heart, lungs and abdomen were normal. Both tibiae showed

marked anterior and lateral bowing. All motions of the left hip were completely limited by muscle spasm and pain. There was no shortening and no crepitus, but complete loss of function of the hip.

X-ray (see Plate I) showed a transverse fracture of the distal extremity of the neck of the left femur. It also showed an area of increased density through which the fracture line passed. There was very marked irregular increase in the density of the bones of the pelvis and femora. The striation stood out very distinctly. Very marked thickening of the shaft of the left femur was also made out. The fragments were in good position. The skull (see Plate II) showed diffuse mottled increase in density characteristic of Paget's disease. The films of the skull were somewhat underexposed and more films were requested. December 2 X-ray confirmed the previous findings. The process apparently involved the lower two-thirds of the dorsal spine, the scapula, the right clavicle and some of the ribs. The bones of the forearm were also involved. The left semilunar, pisiform, unciform, and the right pisiform and trapezium were increased in density. There was slight mottling of the right semilunar. The lower end of the right ulna projected below the articular surface of the radius. It was thought this might be the result of old trauma. There was also visible calcification of the blood vessels of the hand. The lower lumbar spine showed marked involvement.

The urine was normal. The blood calcium was 13.6 milligrams per 100 cubic centimeters of plasma, the inorganic phosphorus 5.9 milligrams. A Wassermann was negative. The other blood findings were not recorded until January. The cerebrospinal fluid gave a negative Wassermann, total protein 35, goldsol 0000000000, one cell.

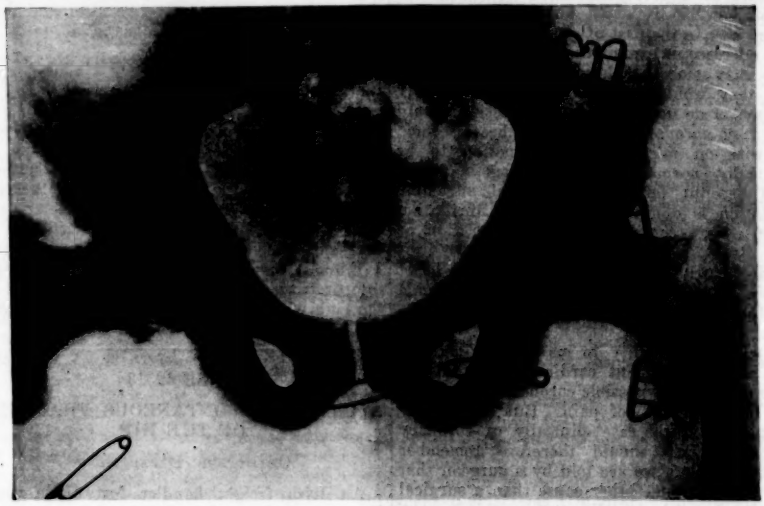


PLATE I. Shows a transverse fracture of the distal extremity of the left femur, and an area of increased density through which the fracture-line passes. There is very marked irregular increase in the density of the bones of the pelvis and femora. The striation stands out very distinctly. Very marked thickening of the shaft of the left femur is made out.

The patient had pain at all times on motion in the left hip, and was unable to raise his head from the bed. There was some eversion of the foot. Sandbags were placed about the leg to prevent external rotation.

The temperature ranged from 96.5° to 102.5°, the pulse from 61 to 132, with a terminal rise to 200. The respirations were usually normal, but on a few scattered days were increased to 30 or 35.

December 7 a biopsy was done. Under gas-ether anesthesia an incision five centimeters long was made over the anterior margin of the left ilium and the tissues were divided down to and including the periosteum. Sections comprising the margin of the ilium extending upward four centimeters from the anterosuperior spine were removed. The wound was closed without drainage. An attempt was made to impact the fracture by use of a mallet. The pathological report was, "Irregular fragments of sclerosed bone

showing on microscopic examination numerous new-formed trabeculae. The marrow is filled with a cell-rich vascular connective tissue and contains occasional multinucleated giant cells." December 15 post-reduction X-rays showed the fragments in good alignment and the fracture line scarcely visible.

December 28 a medical house officer could find nothing in the throat or chest to account for the rise in temperature. The failure of the respirations to rise in proportion to the pulse and temperature was against respiratory infection. He found the patient nauseated and with headache, and thought a gastro-intestinal disturbance might be the cause of the temperature. January 20 the cast was removed. X-rays showed the fracture line present, larger and ragged in outline. Apparent absorption of the neck had taken place. January 29 there was a spherical mass about the size of a large orange, somewhat tender on palpation and semifluctuant, lateral to the great vessels beneath and below Poupart's on the left side. January 30 X-ray showed the old fracture with upward displacement of the shaft. There was no evidence of callus. The pelvis showed well marked changes. . . . Films of both hands (see Plate III) showed thickening of most of the bones. The process apparently involved the long bones of the forearms. The arteries of the hands again showed visible calcification. February 17 the mass in the left thigh was increased in size and was firm and painful. The superficial vessels were dilated. The soft tissues of the entire thigh were somewhat edematous. X-ray (see Plate IV) showed a large area of increased density about the fracture site with some small areas suggestive of calcification. The patient complained of occasional periods of pain at night. The mass in the left groin and upper thigh seemed to be increasing in size. A roentgenological consultant reported, "Deep X-ray will do no harm in this region, and may be of some help."

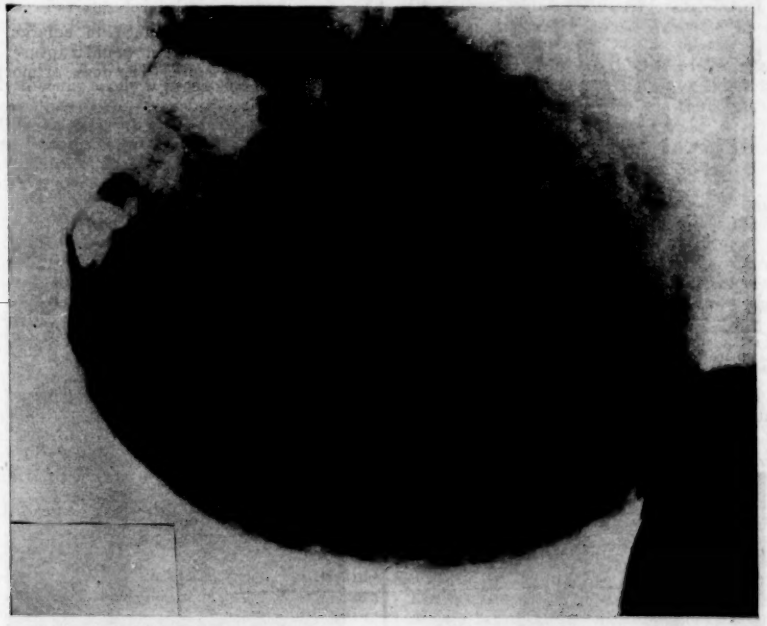


PLATE II. The skull shows diffuse mottled increase in density characteristic of Paget's disease.

February 20 he was sent to the X-ray Treatment Clinic, where a plan was adopted for short wave therapy, a full erythema dose to be directed to the posterior left hip if possible and to an anterior area also, although it was thought that this might be impossible. Three treatments were given February 20, 23 and 24, all to the posterior thigh.

March 17 he had a period of dizziness with paroxysmal extrasystoles. March 22 the swell-

ing was aspirated with a large needle and a small amount of bloody material removed. Pathological report was, "small fragments showing on microscopic examination a tissue too necrotic

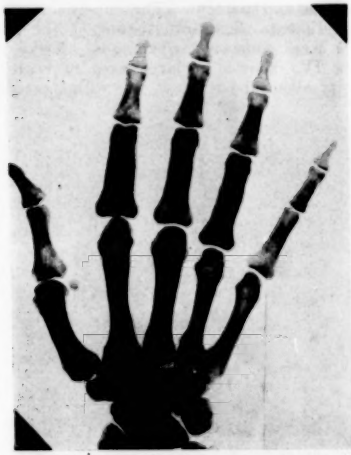


PLATE III. The hands show increased density of some of the phalanges, fairly typical of Paget's disease.

for diagnosis." Contrary to expectation the aspiration hole healed promptly. The tumor mass now increased rapidly in size. The patient had

PLATE IV. February 17. Shows a large area of increased density about the fracture site with some small areas suggestive of calcification.

some pain in the legs and in the region of the left hip.

X-ray April 5 showed rather extensive destruction of the neck of the femur. The destructive process stopped along the intertrochanteric line. The greater part of the greater trochanter was destroyed. There was a line running through the soft tissue suggestive of bone spicules. The chest showed no evidence of disease in the soft tissues. There was apparently a gradually enlarging tumor mass in the right temporo-occipital region. Films of the skull April 27 showed changes similar to those previously described. There was marked thickening of both tables, especially the inner. The process also involved the sphenoid and the first cervical vertebra.

The tumor mass continued to enlarge and gave the impression that it would rupture. The pain became more acute. The doses of morphia

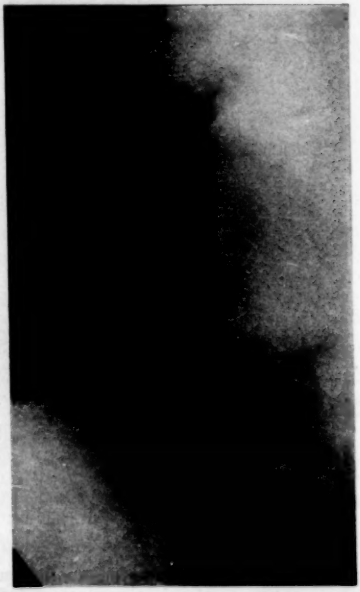


PLATE V. Left femur June 3. Shows extensive destruction of the neck of the femur. The greater part of the greater trochanter is destroyed. There is a line running through the soft tissues suggestive of bone spicules.

sulphate were increased until by the middle of May he was taking seven grains a day. By the end of May the skin was breaking down over the lateral inferior aspect of the midthigh. Zinc oxide dressings were used with no improvement. The area of breaking skin became larger. By the end of June there were pressure sores on the posterior aspect of the thigh. The lateral aspect discharged a small amount of serum growing larger and darker in color.

July 8 the patient had a series of five generalized clonic convulsions without loss of speech or residual palsies. Each seizure lasted

about five minutes. July 10 he had another convulsion lasting eighteen minutes with a residual headache and right hemiplegia. The muscle power was returning that day, but there was still loss of flexion and internal rotation at the shoulder. Transient ankle clonus and Babinski were present. July 12 he had another convulsion lasting nearly three hours, entirely right sided, involving the eyes, face, hands, arm and leg. There was still some power left in the right arm. July 12 a nerve consultant found evidences of right hemiplegia without aphasia. There was Babinski on the right. "With the history of convulsions and the marked peripheral and retinal arteriosclerosis it would seem likely that the cerebral process was one of thrombosis rather than of metastasis." The night of July 13 the patient had another convulsion. There was now total loss of power in the right arm and leg. There was still some in the face, though the lower half was weak. July 17 he died.

Discussion

BY PHILIP D. WILSON, M.D.

This of course is the story of a spontaneous fracture.

That is a significant remark about having to wear a larger hat.

"There was no shortening or crepitus." I think we rarely get crepitus in the early stage of a fractured hip.

In the examination of a patient with fracture I think we are all agreed now that it is a mistake to try to elicit the classical signs of fracture. We try to find out all we can, not neglecting the nervous system, the nerves of the extremity or the circulation of the extremity, without bothering the patient too much, and getting our final impression from X-ray. It is a more humane method. I think the complete testing that we used to do is scarcely justified.

This (Plate I) is the first picture taken, showing the line of fracture through the base of the neck of the femur and an irregular mottled appearance in the bone with some thickening of the shaft. The pelvis shows irregular increase in density. The history of a spontaneous fracture suggests the presence of some disease process in the bone, and in the X-ray film we find confirmation of that suspicion. There is a pathologic process present in the bone concerning which we want to get more information, and that is to be had by complete X-ray examination of the skeleton.

This (Plate II) is a plate of the skull, showing a marked thickening of the bone with a haziness, a cotton-wool appearance, fairly typical of Paget's disease. The lateral view also shows marked thickening and increased density. All of the bones are similarly affected. The humerus is rather large, with very dense cortex. We can scarcely trace the medulla through it. In the hands (Plate III) there is increased density of some of the phalanges, fairly typical of Paget's disease. Some of the other bones are involved also, not so clearly. The forearm, particularly the ulna, shows the increase in density and the irregular mottled appearance. There is general involvement of practically the entire skeleton in this case.

Dr. Cabot: How about that clavicle? I am very much interested in the clavicle in this disease.

Dr. Wilson: I do not think I can say much about the clavicle here. I should think it is probably involved, but I do not think the picture is particularly striking.

Dr. Holmes: I should doubt if we have any evidence that it is. A good deal of that appearance is due to foreshortening.

I should like to mention one or two points with regard to differential diagnosis. The plates of the hands are typical of Paget's disease. One of the things to consider here is metastatic malignant disease, but that practically never involves the hands. We are almost certain it is Paget's disease.

Dr. Cabot: What are the characteristic points in the hands?

Dr. Holmes: The bones, as Dr. Wilson has pointed out, show enlargement. The epiphyses are involved as well as the shaft, and there is bowing of the long bones. It is practically the only disease in an adult in which we get bowing. In metastatic malignancy bones do not bow. Then in Paget's disease we get a change in the bone marking network, which is coarse and irregular, sometimes forming triangles. Some of these plates show it beautifully. Whereas in metastatic malignancy the markings form rings.

Dr. Wilson: It seems to me that here we have a disease beginning in middle adult life following an infection, which may or may not be important, because we do not know the cause of this disease. It has been characterized by a slow and progressive involvement of the long bones with gradual bowing, enlargement of the skull, very little in the way of symptoms,—vague pains. These pains probably are not so much due to any process in the bone itself as to mechanical strain upon the joints from the faulty attitude of the bone and the way the weight is transmitted. With this perfectly characteristic X-ray appearance, showing a generalized involvement of the bones and a spontaneous fracture, I think there can be no question about the diagnosis, and that we are dealing with an osteitis deformans or Paget's disease. Remarks about this disease I will defer until we have finished with the case.

Arteriosclerosis is a common accompaniment

of this process, and it is certainly remarkable to find such well defined arteriosclerosis as is shown by the X-rays in a man of forty-three.

The fracture was not in bad position. In these fractures we expect to get union. Spontaneous fracture or fracture from trauma is not an infrequent finding in Paget's disease, and experience has shown that these fractures will go on as a rule to produce normal callus and unite as rapidly as any normal bone. So in this case whether he had Paget's disease or not, the important thing was to treat his fracture and get the fragments in position. Incidentally we wanted to get as much information as we could about the bone disease. We had to give him an anesthetic, and advantage was taken to excise a small portion of the iliac crest for pathological examination. That was surgical curiosity, and I do not believe it did the patient any harm. I do not believe it did him any good.

We reduce these fractures of the hip by swinging them out in abduction and internal rotation, which tends to force the fragments together, and by fixing them with plaster. In some of these cases impaction can be brought about by using a mallet. Dr. Cotton has done that in a good many cases. We anticipated a little difficulty in this case, so we used the mallet.

Post-reduction X-ray showed good position. In fact the fracture line is scarcely visible. There is good approximation of the fragments.

He had very little reaction after the operation, and ran along normally until the 22nd day, when the temperature rose to 101.6°, the pulse to 125. But it was of short duration, and following that the temperature ran along fairly normally again. The cast was on about seven weeks. That is a little early to remove it, but he was having some discomfort, and I think that was the reason.

In January for the first time it was noted that a mass was developing over the front of the hip. The X-ray shows great change. Part of the neck of the femur has disappeared. There is a large area of destruction appearing. It is more than simple absorption. At any rate we have non-union. By January 30 we are dealing with a tumor supervening in the region of the fracture, apparently originating in the bone, at least if the destructive process is any guide.

Dr. Cabot: What was this mass which appeared January 29?

Dr. Wilson: Of course that is the question. We have no definite clue except for the fact that very frequently we find sarcoma occurring as a complication in osteitis deformans. The figures on that are rather confusing. I found figures by one man stating that it occurred in twelve per cent. of sixty-two reported cases. I think Dr. Wolbach says something like seven per cent. Practically all museums have specimens of sarcoma developing on the background of an osteitis deformans. We do not know what this tumor is in this particular case. We only know that there is a destructive process in the bone, and we suppose that it is a sarcoma because it arises in the bone.

Dr. Cabot: "Fluctuant" is a queer statement. It is not an inflammatory mass, and there is nothing about the picture to suggest an inflammatory condition.

Dr. Wilson: What do the extrasystoles mean?

Dr. Cabot: That does not mean anything characterizing any disease of the heart. Anybody can have it.

Dr. Wilson: New X-rays were made in February which give a little better idea of the mass, and there is a little appearance of calcification in these plates too. (Plate IV.) They were looking for pulmonary metastases, but none was found. There was no change noted in the plates of the skull.

Actually I think the case record is wrong in describing the location of the tumor of the head. We notice that the tumor mass in the skull is said to be located on the right side of the skull. I remember it as being on the left, and it is only if it were on the left that it could explain the right-sided hemiplegia.

I think that the diagnosis here is perfectly clear, with a tumor growing at such a rate of speed that it was only five months from the time we first saw it appear until it reached this stage where it was destroying the entire upper end of the femur. In the plate taken in June (Plate V) we can get an idea of the enormous size of the tumor. There is nothing of the neck of the femur left and nothing of the upper portion of the shaft. There is no doubt that we are dealing with a very malignant tumor, probably a sarcoma supervening on a Paget's disease and at the site of a spontaneous fracture.

Dr. Cabot: Do you suppose the tumor was there before the fracture?

Dr. Wilson: In comparing the X-rays of the two hips I think you will find exactly the same appearance. The neurologist thought that thrombosis was the cause of the hemiplegia. With the presence of a skull tumor which I am going to say was left-sided, probably enlarging internally, I should think we have enough to explain these pressure symptoms, his convulsions and the right-sided hemiplegia. So my diagnosis would be osteitis deformans, and he may have had a terminal septicemia I suppose, from the fact that he had a large wound over his thigh with this sloughing tumor.

Differential diagnosis of osteitis deformans I think would bring up the question of syphilis, as Dr. Holmes mentioned, and certainly the X-ray appearance of syphilis is entirely differ-

ent. It is a periosteitis and I do not believe could possibly give this appearance.

We have other disturbances of bone metabolism. Osteitis fibrosa is a disease only vaguely understood. I think many of us do not speak about the same disease when we mention that name. But if we stick to Von Recklinghausen's definition we mean a disease characterized by the formation of cysts in the ends of the bones, and we differentiate two types, the single cysts, and multiple cysts, and on histological examination we find a tissue where the marrow has been

PLATE VI. Right and left femur at necropsy. The remains of the head of the left femur is seen above the shaft.
DR. OSCAR RICHARDSON.
Photograph by Louis M. Adams.

replaced by a connective tissue rich in giant cells, and that there is a little new formation of bone.

Osteomalacia is a disease which is more apt to occur in women, especially after pregnancy, or in certain regions of the world where certain dietetic conditions prevail, and is characterized by lack of density of the bones. Dr. Holmes I hope will say something about the special X-ray appearance. It seems to me that the differentiation in this case is perfectly easy.

A PHYSICIAN: Did you say there were no other metastases except to bones?

DR. WILSON: None was found.

A PHYSICIAN: And the Wassermann?

DR. WILSON: The Wassermann was negative, and Wassermann on the spinal fluid was negative. There have been many attempts made to explain Paget's disease as due to syphilis, but I think the majority of us agree that it cannot be.

DR. CABOT: Have we ever had a Paget's disease necropsy here before?

DR. RICHARDSON: I think we have had one or two.

DR. WILSON: From the appearance of the gross specimen (Plate VI) it may be seen that the entire upper third of the femur had been destroyed, leaving only the articular surface. On section the femora showed marked increase in the width of the cortex, in places scarcely any medullary canal persisting. In the cortex there may be seen irregular areas of sclerotic bone, bones alternating with areas of bone that are extremely vascular, newly formed. The histological picture is that of a degenerate form of osteogenic tissue. Absorption of the old bone is proceeding, but the new bone that is replacing it is a poor substitute, merely osteoid tissue. The bone trabeculae are very thin, the cortex is very distorted, and one may imagine that it would be a relatively slight step for it to be transformed into malignant sarcomatous tissue.

DR. CABOT: Would anybody today call this disease inflammation, "itis," "osteitis"?

DR. RICHARDSON: I should not think so.

DR. CABOT: It is a degenerative process?

DR. RICHARDSON: There is softening and resorption of the bone, then excessive proliferation, resulting in a mixture of soft bone-like and fibrous tissue, and in cases like this one the development of a sarcoma in places.

DR. WILSON: It is very interesting that the first case seen by Paget, which was observed over a period of nearly thirty years, died of a tumor developing in the radius.

A PHYSICIAN: Was the fracture of the hip due to sarcoma or to the osteitis?

DR. WILSON: It had been weakened by the osteitis. This bone is extremely brittle and has lost all its elasticity. We could detect no evidence of sarcoma at entrance.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Osteitis deformans (Paget's disease), generalized.
Fracture of the neck of the left femur, pathological.
Sarcoma of the left femur.
Thrombosis of cerebral artery.
Hemiplegia.
General arteriosclerosis.
Excision of a portion of the crest of the ilium for diagnosis.

DR. PHILIP D. WILSON'S DIAGNOSIS

Osteitis deformans.
Osteogenic sarcoma of the femur with metastasis to the skull.
Arteriosclerosis.
Septicemia.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

Osteitis deformans.
Osteogenic sarcoma of the left femur with metastases in skull and brain.

2. *Secondary or terminal lesions*

Slight thrombosis of the longitudinal sinus.
Area of hemorrhage, left cerebral hemisphere.
Edema of the lungs.
Hypostatic pneumonia.
Soft hyperplastic spleen.

3. *Historical landmarks*

Chronic pleuritis.

DR. RICHARDSON: The X-rays show the condition of the bones very well. The mass in the region of the left hip and thigh was of great size. The thigh was greatly swollen, and the surface of the mass necrosed and burst open in places. I shelled out the material from the center of the mass, which consisted of firmer and softer boggy necrotic tissue infiltrated with old blood-clot-like material. This material extended down to the region of the bone, where it was mixed up with bony fragments and the disintegrated remains of the upper third of the femur. (See Plate V).

Coming to the skull, which is another interesting point, the meninges, the vessels of Willis, middle ears, and sinuses were negative except for some slight thrombosis of the longitudinal sinus. The skull generally was very thick and the bone woodeny. The frontal region was ten millimeters thick, the temporal region up to twelve millimeters thick, and the occipital region up to fifteen millimeters thick. In the left posterior parietal region the skull on its outer surface presented a slightly bulging area two and a half centimeters across which was found to be the outer surface of an irregular mass of new-growth-like tissue about 5 by 4 by 2 centimeters, which was adherent to the necrosed wall of that portion of the skull, through which it protruded. Along its inner margin this mass pressed on the longitudinal sinus and the brain with slight thrombosis of the sinus for a short distance. There was an area of hemorrhage two and a half by two centimeters closely associated with the base of the new-growth-like mass where it had pressed into the brain on that side. The pineal and pituitary glands were negative.

DR. CABOT: How about arteriosclerosis? Was there any more than one would expect at his age?

DR. RICHARDSON: No. In the vessels I examined there was not. Of course coming down into the thigh, the vessels there were all lost in the sloughing mass and there may possibly have been some in those. But the great vessels generally were negative.

DR. CABOT: And the heart and aorta did not show anything?

DR. RICHARDSON: No, they were frankly negative. There was some hypostatic pneumonia in the lower lobes of the lungs.

DR. WILSON: It seems to me that clears up everything except the question, what is Paget's disease?

THE PREVENTION OF HUMAN RABIES THROUGH THE TREATMENT OF ALL BITTEN BY RABID ANIMALS

THE Bureau of Preventable Diseases reports some alarming figures relative to the rabies situation in the City of New York. Not only has there been a most disturbing increase in the number of rabid dogs in New York City and in the number of bites which these animals inflict, but it has been demonstrated that only about fifty per cent. of those bitten by rabid animals accept antirabic treatment.

There have been 449 persons bitten in 1926.

It is of course true that only about 16 per cent. of the human beings bitten by rabid animals, if not treated, develop rabies. But the antirabic treatment reduces this figure to 0.5 per cent.

Fuming nitric acid is the best cautery, and is helpful if used within forty-eight hours after the bite. The acid should be applied on the point of a tapered glass-rod or drop by drop from a capillary pipette, so that the amount may be carefully controlled. Contact with bony, cartilaginous or bloodless parts should be avoided, if possible. To these parts apply pure carbolic acid and the fuming nitric acid to the other tissues adjacent. Such tissues heal well, after the use of nitric acid.—*Excerpt from The Bulliten of the New York City Department of Health.*

THE BOSTON
Medical and Surgical Journal

Established in 1828

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

For three years HOMER GAGE, M.D., *Chairman*
EDWARD C. STREETER, M.D.
EDWARD W. TAYLOR, M.D.

For two years WILLIAM H. ROBEY, JR., M.D.
ROGER I. LEE, M.D.
ROBERT B. OSGOOD, M.D.

For one year JOHN W. BARTOL, M.D.
HORACE D. ARNOLD, M.D.
CHANNING FROTHINGHAM, M.D.

EDITORIAL STAFF

DAVID L. EDSALL, M.D.
REID HUNT, M.D.
FRANCIS W. PEABODY, M.D.
JOHN P. SUTHERLAND, M.D.
GEORGE R. MINOT, M.D.
FRANK H. LAHEY, M.D.
STEPHEN RUSHMORE, M.D.
HANS ZINSSER, M.D.
BENJAMIN WHITE, PH.D.
HENRY R. VIETS, M.D.
ROBERT N. NYE, M.D.
SHIELDS WARREN, M.D.

WALTER P. BOWERS, M.D., *Managing Editor*

ASSOCIATE EDITORS

GEORGE G. SMITH, M.D.
WILLIAM B. BREED, M.D.
JOSEPH GARLAND, M.D.

SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, $7.56 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 126 Massachusetts Ave.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The Boston Medical and Surgical Journal, 126 Massachusetts Ave., Boston, Mass.

Christmas

God rest you merry gentlemen
And may you see the star
That lights the way for Christmas Day
On holy hills afar.

Above the ancient manger town
It gleams with radiance rare,
And shepherds see the wise men three
Bow down to greet it there.

And high above a Christian world,
Still splendid now, as then,
Through all these years of hopes and fears
It shines for Christian men.

Shine on, bright star, and light the road
Aspiring man must tread;
That one high beam shall reign supreme
And always glow ahead.

God rest you merry gentlemen
Upon this Christmas Day.
The star is bright your path to light;
Let nothing you dismay.

THE clock that marks the years has seen its hands turn round a full circle on the dial again, and again the friendly season is here when men's thoughts are united in one common thought; when a kindly spirit prevails that is at other seasons too often lacking. At other times we are drawn apart; our interests are more our own than common ones; our aims, we must admit without pride, are largely selfish. Different creeds we cleave to, or none at all, and many who let their thoughts stray towards Christianity must doubt the power it exerts over a twentieth century world.

We must leave to other, better adapted pens the writing of sermons; the lesson to be learned is that any great idea capable of influencing men's thoughts and firing their imaginations and creating such an attitude of goodwill as prevails at this time must still be a power in the world.

Stevenson has written of Medicine, "There are men and classes of men that stand above the common herd: the soldier, the sailor and the shepherd not infrequently; the artist rarely; still more rarely the clergyman; the physician almost as a rule. He is the flower (such as it is) of our civilization; and when that stage of man is done with, and only remembered to be marvelled at in history, he will be thought to have shared as little as any in the defects of the period, and most notably exhibited the virtues of the race."

Such a passage cannot be read without a blush of shame and regret that it is not better deserved. We all, or nearly all of us, cherish our ideals of professional service whether rendered at the bedside or in the laboratory or among the students in whom we would kindle, had we the grace, the same fire that we wish might burn more brightly in ourselves.

No; we are not so far above the common herd as sentiment has sometimes placed us; although we have seen the goal we have not reached it, but at this season it is more clearly in our sight than ever, and it is attainable. We must follow the gleam. We must infuse the entire year with the spirit of Christmas.

CLARIFYING THE TUBERCULOSIS SITUATION IN BOSTON

AFTER a considerable period of suspicion and distrust with respect to the management of Boston's tuberculosis problems, some persons took it upon themselves to learn the facts relating to the situation. Very little study led to the conclusion that there was reasonable ground for criticism of the methods employed by the city. Any health problem involves definite civic responsibilities, but how to deal with a situation which might bring to the surface evidence of personal and institutional inefficiency is a task

which is distasteful and not infrequently creates suspicion of the motives of a critic. Then too it is quite possible at times to do more harm than good in an attack on methods of treatment which if left alone would in time be remedied.

We believe that it was only after certain persons were convinced that the tuberculosis problems of Boston were not being dealt with in a constructive way and that no adequate plans for betterment were under consideration certain persons felt that the time had come for a demonstration of inadequacy in order to bring about changes which would put Boston above criticism.

Individual comments and suggestions without demonstrable facts would only have made a bad matter worse and diplomacy necessitated the study of the conditions by an open minded expert and a non official semi public health agency, hence it was logical that the Boston Tuberculosis Association should employ a suitable person to tabulate the facts and make recommendations.

Dr. Murray P. Horwood was employed and his report was submitted to a committee which studied the findings and recommendations with care and suggested such modifications as appeared to the committee to be wise.

This report was submitted to the Mayor of Boston and upon reference to the officials in charge of the tuberculosis work very naturally led to pointed criticism of the report and exceptions to the recommendations.

The Mayor found himself confronted by different opinions relative to the quality of the service rendered but he as a layman was unable to form conclusions.

His delay in taking definite action may be excusable because a non medical mind could not be expected to separate the wheat from the chaff.

He recognized the need for action and imported a recognized expert in the person of Dr. Haven Emerson to go over the ground. According to newspaper accounts of Dr. Emerson's report to the Mayor the main features of Dr. Horwood's report were endorsed and definite recommendations presented. Reading the two reports there is much unanimity of opinion expressed by these experts.

The last step to date in this movement took the form of a meeting held December seventeenth under the auspices of the Public Health League of Boston at 14 Beacon Street.

The meeting was called to order by Dr. John Bartol, President of the League, who in his opening remarks left the impression that the League had some influence in the selection of Dr. Haven Emerson by the Mayor as an expert to make an independent study of the situation and report thereon. Dr. Emerson was introduced to an audience composed of officials of the city, representatives of various public health organizations and others who are interested in Boston's affairs.

Dr. Emerson made running comments on the conditions which he found in his study, reading parts of his report of fifty pages during the address. In speaking of tuberculosis as it existed in this country, he stated that Boston and Chicago are open to the severest criticism among the largest cities of the United States and are now worse off than formerly. He recommended that the present Board of Trustees of the Mattapan Hospital should be discontinued, and the problem be dealt with by the Boston Department of Health with a division of tuberculosis in charge of a well-trained and experienced executive. The defects of the present system were due to a faulty structure and a plan should be evolved which would bring co-operation between official and voluntary organizations.

In order to bring about greater efficiency he felt that the officer in charge of the work should be represented on the Board of Trustees of the City Hospital and that the Trustees of the City Hospital should take on the institutional care of tuberculosis patients. Under the present system the City Commissioner of Health has not the adequate authority which should be conferred on him.

He deplored the unnecessary use of police power in dealing with the unruly tuberculosis patient who might be a menace and that the psychology of dealing with this danger may consist in making the hospital sufficiently attractive so that the otherwise unruly patient would come to prefer hospitalization rather than freedom which might mean danger to others. Power of control should be a last resort but the dangerous patient must be taught how to behave.

A very pointed criticism of the Boston doctors could be based on the low ratio of reported cases of tuberculosis as compared with the deaths. In Boston the number of deaths due to tuberculosis indicated that at least twice as many cases should have been reported by the physicians and the physicians must be made to report these cases. Not less than five cases should be reported for every death per annum.

Condemnation of the present out-patient clinic was made and the recommendation offered that decentralization and the creation of clinics in close proximity to the homes of patients should be effected. The criticism of the tuberculosis clinic in Boston was scathing, for it neither appears attractive nor does it function properly. He deplored the waste of time and energy of visiting nurses attached to the central clinic who are trying to serve a large area. He felt that more intensive education of doctors and nurses is needed and that all doctors

and all nurses should have contact with tuberculosis patients. In this respect, he felt that the Boston Tuberculosis Association should study the situation more definitely.

So far as hospital accommodations are needed, there should be one hundred more beds and a separate building near the present hospital should be erected and equipped but under a common management. Preventorium work, incipient and advanced cases should be handled as features of one problem.

Under present conditions and management, repeated and continuous exposures by untreated or improperly treated patients will defeat efforts at reduction of cases. There are not enough follow-up visits of nurses.

Referring again to the Mattapan Hospital he felt that the care of the patients should be carried on by well people and not by other patients.

In closing his address, Dr. Emerson paid a tribute to the long and valuable service rendered by Dr. J. J. Minot.

One may seriously question the apparent complacency of those formerly in charge of Boston's tuberculosis problems and further wonder why the Trustees in charge of the Sanatorium should have omitted to recommend certain very obvious changes, or at least asked for authority to have the conditions investigated by an independent authority.

We trust that the uncomfortable feelings engendered will be smoothed away and everybody will unite in support of plans which will be evolved to meet the needs of existing conditions. Local public health workers will follow developments with interest.

CONGRATULATIONS TO THE HERALD

The *Boston Herald* for December 14, 1926, announces the campaign of the Federal Trade Commission to war on fake medical advertisemen.s. This campaign, according to the announcement, will be a determined fight to exterminate fake advertisements of "anti-fat" remedies, patent medicines for incurable diseases, beauty lotions, creams, soaps and other cosmetics, "health belts" and industrial schools practising fraud.

W. E. Humphrey, member of the commission, estimated that such fraudulent advertisers obtain more than $500,000,000 annually from the public, and made the statement that "there is no viler class of criminals known among men than those publishers and advertisers." Mr. Humphrey further stated that publishers who allow such fraudulent advertisements to appear in their publications "should be held equally responsible with the crooked advertisers."

The publication of this Associated Press despatch by the *Herald* must indicate either that the *Herald* is in sympathy with the campaign or that news and a newspaper's advertising policy have nothing in common. It is sincerely to be hoped that the first assumption is true, but past experience has led us to believe that it cannot be so unless a change of heart has taken place, for in the past and certainly until very recently, the *Herald* has occupied a notorious position among newspapers for the baseness of its medical advertising.

The issue of the *Herald* in which this news item appears is singularly free from the type of advertising that it so naïvely flagellates. True, certain so-called remedies not approved by the Council on Pharmacy and Chemistry are retained but some of the more objectionable advertisements are temporarily, at least, missing from the pages.

Perhaps the efforts of the Journal and other well wishers to humanity have availed something; the *Herald* has had a change of heart and has commenced the purging of its pages. If so, we offer the editors our heartiest congratulations. May the good work continue until it is completed.

THIS WEEK'S ISSUE

CONTAINS articles by the following authors:

SCUDDER, CHARLES L., A.B., Ph.B., M.D. Harvard Medical School, 1889, F.A.C.S. Consulting Surgeon Massachusetts General Hospital, formerly Assistant Professor of Surgery Harvard Medical School, member American Surgical Association, Chairman Fracture Committee American College of Surgeons. His subject is "The Operative Treatment of Fractures." Page 1187. Address: 144 Commonwealth Ave., Boston.

WASHBURN, FREDERIC A., A.B., M.D. Harvard Medical School 1896, Director Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary, President Medical Superintendents' Club, Past President American Hospital Ass'n. His subject is "Remarks on the History of the Old Surgical Amphitheatre." Page 1193. Address: Massachusetts General Hospital, Boston.

WHITE, LEON E., A.B.; M.D. Dartmouth Medical School 1893. Surgeon in Otolaryngology Massachusetts Eye and Ear Infirmary, Otologist Massachusetts General Hospital. His subject is "The Influence of Negative Pressure in the Sphenoid on the Optic Nerve." Page 1195. Address: 390 Commonwealth Ave., Boston.

LENNOX, WILLIAM G., A.B.; M.D. Harvard Medical School 1913. Research fellow in neuropathology Harvard Medical School, Assistant

in Medicine Harvard Medical School and Thorndike Memorial Laboratory, Member American Society for Clinical Investigation and Association for Research in Nervous and Mental Diseases. Address: Harvard Medical School. Associated with him is

WRIGHT, LESLIE H., M.D. University of Vermont 1918. Formerly Assistant Physician Connecticut State Hospital and formerly Pathologist to Monson State Hospital, now Assistant Superintendent Peter Bent Brigham Hospital, Secretary and Treasurer New England Hospital Association. Address: Peter Bent Brigham Hospital, Boston. They write on "Comparative Effects of Borotartrate and Luminal on the Seizures of Epilepsy." Page 1199.

BARLOW, ROY A., B.S.; M.D. University of Michigan 1914, F. A. C. S., Associate Otolaryngologist Mayo Clinic 1916-1917, Assistant Professor of Otolaryngology University of Minnesota 1921-1922, Fellow American Laryngological, Rhinological and Otological Society. His subject is "Halle Operation for Ozena." Page 1202. Address: Madison, Wis.

LEGISLATIVE NOTE

A BILL FOR LEGISLATIVE ACTION

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Twenty-Six

AN ACT RELATIVE TO REPORTS OF TREATMENTS OF WOUNDS CAUSED BY FIREARMS

Be it enacted by the Senate and House of Representatives in General Court assembled, and by the authority of the same as follows:

SECTION 1. Every physician attending or treating a case of bullet wound, gunshot wound, powder burn, or any other injury arising from or caused by the discharge of a gun, pistol or other firearm, or whenever such case is treated in a hospital, sanitarium or other institution, the manager, superintendent or other person in charge shall report such case at once to the police authorities of the city or town where such physician, hospital, sanitarium or institution is located. The provisions of this section shall not apply to such wounds, burns, or injuries received by a member of the armed forces of the United States or the Commonwealth of Massachusetts while engaged in the actual performance of duty.

Any violation of this act shall be punished by a fine of not less than fifty nor more than one hundred dollars.

MISCELLANY

THE CANCER PROBLEM

THE daily papers announce that W. L. Saunders of New York has offered a prize of $50,000 to any person or group of persons who may discover what human cancer is and how it can be prevented and another prize of the same amount for a cure of cancer.

The offer was made known at a dinner in the interest of the American Society for the control of Cancer. The offer holds good for three years. Mr. Saunders is a director of the Federal Reserve Bank of New York.

We predict that this prize will not be awarded to Koch.

BEWARE OF THE TYPHOID CARRIER

IT is reported that the State Department of Public Health has traced the cause of forty cases of typhoid fever in Lincoln and Concord to a typhoid carrier who milked cows on a certain farm.

This carrier was reported from Connecticut as having migrated to Massachusetts but was not found in the town where it was expected that he would secure work. He was subsequently found in Lincoln.

ERRATA

IN the article by Paul D. White, M.D., in the issue of December 16, 1926, the last two lines and foot note in the second column at bottom of page 1146 belong at the end of the first column on the same page.

Near the bottom of the second column, page 1147, *ausculatory* should have appeared as *auscultatory*.

Also note that the name Graham *Steele* which appears in this article should be Graham *Steell*.

THE SEAL SALE

ONE hundred and ten thousand dollars has been raised so far in the Christmas Seal Campaign, now going on throughout the State, for the prevention of tuberculosis in 1927. This is a little less than half the total amount raised in 1926. The quota for this year is $250,000.

Dr. Kendall Emerson of Worcester, President of the Massachusetts Tuberculosis League, in reviewing the progress of the Seal Sale, said recently, "We have every reason to expect that the annual Christmas Seal Sale, which for the past eighteen years has been carried on successfully for the prevention of tuberculosis, is to equal the sales of previous years. One peculiar phenomenon, however, has been called to my attention during the course of this Seal Sale and that is the failure of the people who pur-

chase the Christmas Seals to use them as widely as in previous years on Christmas packages, letters and greeting cards. This is true in many parts of the Commonwealth and it is the hope of the tuberculosis associations that during the remaining days before Christmas people will beautify their greetings and gifts by an extensive use of this year's Seals.

"The 1926 Seals are unusually decorative and add considerably to the attractiveness of anything to which they are affixed. The Seals bear the picture of three medieval troubadours singing Christmas carols. We hope that in the few remaining days before Christmas every letter, greeting card and package sent through the mails will bear not only one but many Seals.

"The tuberculosis associations are planning for extensions of their work in the prevention of disease, especially among children, in 1927."

RECENT DEATHS

MATHES—Dr. Roy Wentworth Mathes died at his home in Lynn, December 14, 1926, following a ten days' illness of pneumonia.

Dr. Mathes was a native of Gorham, N. H., where he was born in 1883. He studied at Phillips Exeter Academy, and entering Dartmouth Medical School, was graduated in the class of 1906. He went to Lynn that same year and associated himself with Dr. Charles H. Bangs and later with Dr. Arthur E. Joslyn. In 1913 he was appointed Eastern district physician under former Mayor George H. Newhall. During the World War Dr. Mathes saw service in the Navy, holding the rank of lieutenant.

He was a member of the Massachusetts Naval Reserve, the American Medical Association, the Massachusetts Medical Society and the Lynn Medical Fraternity. He served two terms at the Contagious Hospital in Lynn, and three years as school health inspector. He belonged to the Oxford Club, Alpha Kappa Fraternity of Dartmouth, and was identified with the Central Congregational Church, where he was active in the men's class.

Dr. Mathes is survived by his wife, Madeline Mathes, who is a member of the Lynn School Board; three children, a brother and a sister.

BINNEY—Dr. George Hayward Binney died at his home in Boston, December 4, 1926, aged 40.

He was the son of the late George H. Binney and Mrs. Edith Marsh Binney and was born in Boston. He attended the Noble & Greenough School and graduated from Harvard with the class of 1908. In 1914 he was graduated from the Harvard Medical School. He had since been a practicing surgeon, with offices at 45 Bay State Road.

He is survived by his widow, Mrs. Susan Appleton Binney, three daughters and three sons.

CORRESPONDENCE

THE LATE DR. ELLIOTT COUES ON THE ALCOHOL QUESTION

Mr. Editor:

The following forceful and graphic remarks are from "Field Ornithology" and were addressed by Dr. Coues to the ornithologist in the field:

"Stimulation. It should be clearly understood in the first place that a stimulant confers no strength whatever; it simply calls the powers that be into increased action at their own expense. Seeking real strength in stimulus is as wise as an attempt to lift yourself up by the boot-straps. You may gather yourself up to leap the ditch, and you clear it, but no such muscular energy can be sustained; exhaustion speedily renders further expenditure impossible. But now suppose a very powerful mental impression be made; say the circumstance of a succession of ditches in front, and a mad dog behind; if the stimulus of terror be sufficiently strong you may leap on till you drop senseless. Alcoholic stimulus is a parallel case, and is not seldom pushed to the same degree. Under its influence you never can tell when you *are* tired; the expenditure goes on, indeed with unnatural rapidity, only it is not felt at the time, but the upshot is you have all the original fatigue to endure and to recover from, *plus* the fatigue resulting from overexcitation of the system. . . . There is no use in borrowing from yourself and fancying you are richer. . . . Stimulation is a draught on vital capital, when interest alone should suffice; it may be needed at times to bridge a chasm, but habitual living beyond vital income infallibly entails bankruptcy in health. . . . The three golden rules are,—never drink before breakfast, never drink alone, and never drink bad liquor. . . . Serious objections for a naturalist, at least, are that science, viewed through a glass, seems distant and uncertain, while the joys of rum are immediate and unquestionable, and that intemperance, being an attempt to defy certain physical laws, is therefore eminently unscientific."

Very truly yours,

Wm. Pearce Coues, M.D.

December 14, 1926.

A WARNING TO PHYSICIANS

December 16, 1926.

Editor, Boston Medical and Surgical Journal:

With this I send a page from the December 6 issue of the *Insurance Age-Journal*.

It contains a warning to physicians which seems of importance.

Yours very truly,

David N. Blakely.

The clipping contains a copy of a letter to the President of the Postal Life Insurance Company written by Harold J. Taylor, counsel of the Massachusetts Insurance Department, in which it is set forth that the actuary of the Department of Insurance has received a circular soliciting him to take a policy of life insurance with the Postal Life Company. It is stated in the circular that this company has physicians in nearly every community who conduct physical examinations of applicants for insurance.

Mr. Taylor quotes Section 160 of Chapter 175 of our General Laws, which provides that whoever, for a person other than himself, negotiates or acts or aids in any manner in the negotiation of a policy of insurance issued by an unlicensed foreign company is punishable by a fine of not less than $100 or more than $500. He comments as follows:

"This section is very sweeping in its terms, and any person doing any act in this Commonwealth which is proximately connected with the issue of such a policy or which is a link in the chain of events culminating in the issue of such a policy is liable to the penalties of said section. The company apparently will not insure a person who has not passed a medical examination which is therefore a necessary step in the negotiations between it and the applicant looking to the issue of a policy. The act of the physician who examines the applicant to whom a policy is thereafter issued is, indubitably, proximately connected with its issue, that is, he has acted

or aided in some manner in the negotiation of the policy in violation of said Section 160.

"It is true that a resident of this or any other State has a right, which cannot be abridged by State legislation, to accept a policy issued by an unlicensed foreign company, and that such a company has a right to solicit and transact business through the medium of the mail. This statute does not affect those rights. It does, however, prohibit any person from acting in this Commonwealth, either as the agent of the applicant or insured or of the unlicensed company and has been declared to be constitutional by the Supreme Courts of this Commonwealth and of the United States.

"This matter may not interest the company but we feel that it should be called to your attention so that you may know that any doctors examining applicants in this State expose themselves, perhaps unknowingly, to arrest and criminal prosecution under this section.

"Section 3A of said chapter imposes upon this department the mandatory duty of enforcing said chapter and if satisfactory evidence is forthcoming from any source that any physician acting as aforesaid has examined a person to whom you have issued a policy, appropriate action will be in order."

It would be unfortunate if any physician in Massachusetts should be prosecuted.

CONNECTICUT DEPARTMENT OF HEALTH

Morbidity Report for the Week Ending December 11, 1926

Diphtheria	37	Conjunctivitis, infectious	3
Last week	32	German measles	2
Diphtheria bacilli carriers	14	Influenza	5
Scarlet fever	52	Mumps	12
Last week	58	Pneumonia, lobar	32
Measles	39	Septic sore throat	3
Last week	69	Trichinosis	1
Whooping cough	30	Tuberculosis, pulmonary	15
Last week	33	Tuberculosis, other forms	1
Typhoid fever	2	Gonorrhea	9
Last week	0	Syphilis	14
Bronchopneumonia	20		
Chickenpox	119		

CASES REPORTED TO THE MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH FOR THE WEEK ENDING DECEMBER 11, 1926

Anterior poliomyelitis	2	Ophthalmia neonatorum	29
Chickenpox	416	Pneumonia, lobar	95
Diphtheria	104	Scarlet fever	324
Dog-bite requiring anti-rabic treatment	19	Septic sore throat	6
		Suppurative conjunctivitis	2
Encephalitis lethargica	1	Syphilis	16
Epidemic cerebrospinal meningitis	1	Trachoma	2
		Tuberculosis, pulmonary	84
German measles	8	Tuberculosis, other forms	11
Gonorrhea	42		
Influenza	9	Tuberculosis, hilum	3
Measles	56	Typhoid fever	6
Mumps	166	Whooping cough	148

RESUME OF COMMUNICABLE DISEASES FOR MASSACHUSETTS

November, 1926

GENERAL PREVALENCE

The common communicable diseases which showed an increase over last month were chickenpox, diphtheria, mumps, scarlet fever and whooping cough.

	Nov., 1926	Oct., 1926	Nov., 1925
Chickenpox	1,232	420	805
Diphtheria	418	291	351
Mumps	599	269	165
Scarlet fever	1,191	729	781
Whooping cough	442	322	718

RARE DISEASES

Anterior poliomyelitis was reported from Auburn, 2; Boston, 3; Brockton, 5; Cambridge, 1; Danvers, 1; Fall River, 1; Greenfield, 1; Haverhill, 1; Leominster, 1; Malden, 1; Medford, 1; Newton, 1; Pittsfield, 3; Revere, 1; Somerville, 1; Worcester, 2; total, 26.

Anthrax was reported from Haverhill, 2.

Dog-bite requiring anti-rabic treatment was reported from Cambridge, 1; Chelmsford, 2; Everett, 1; Franklin, 1; Lowell, 7; Mansfield, 1; Newton, 1; Revere, 3; total, 17.

Encephalitis lethargica was reported from Danvers, 1; Fairhaven, 1; Worcester, 1; total, 3.

Epidemic cerebrospinal meningitis was reported from Boston, 1; Cambridge, 1; Westfield, 1; total, 3.

Malaria was reported from Newton, 1.

Pellagra was reported from Boston, 1.

Septic sore throat was reported from Boston, 7; New Bedford, 1; Newburyport, 1; Weymouth, 1; total, 10.

Trachoma was reported from Boston, 5; Fall River, 1; Plymouth, 1; total, 7.

Trichinosis was reported from Gardner, 1.

DISTRIBUTION

All Communicable Diseases

	Nov., 1926	Nov., 1925
Total cases (all causes)	5,914	8,090
Case rate per 100,000 population	140.2	194.6

Certain Prevalent Diseases

Diphtheria	Nov., 1926	Nov., 1925
Total cases	418	351
Case rate per 100,000 population	9.9	8.4

Cases in cities and towns that have noticeably exceeded their median endemic indexes*:

Salem	24	Spencer	14

Measles	Nov., 1926	Nov., 1925
Total cases	161	3,321
Case rate per 100,000 population	3.8	79.9

Cases in cities and towns that have noticeably exceeded their median endemic indexes*:

Medway	24

Scarlet Fever	Nov., 1926	Nov., 1925
Total cases	1,191	781
Case rate per 100,000 population	28.2	18.8

Cases in cities and towns that have noticeably exceeded their median endemic indexes*:

Franklin	14	Quincy	38
Lynn	31	Watertown	19
Needham	21		

Tuberculosis, Pulmonary	Nov., 1926	Nov., 1925
Total cases	386	388
Case rate per 100,000 population	9.2	9.3

Tuberculosis, Other Forms	Nov., 1926	Nov., 1925
Total cases	62	29
Case rate per 100,000 population	1.5	.7

	Nov., 1926	Nov., 1925
Typhoid Fever		
Total cases	44	35
Case rate per 100,000 population	1.0	.8

Cases in cities and towns that have noticeably exceeded their median endemic indexes*:

Belmont	21	Ashburnham	9

	Nov., 1926	Nov., 1925
Whooping Cough		
Total cases	442	718
Case rate per 100,000 population	10.5	17.2

*The median endemic index is obtained by arranging in arithmetical sequence the monthly totals of reported cases for the past five years and selecting the middle figure.

NEWS ITEMS

LECTURE ON PUBLIC HEALTH LAW—Mr. James A. Tobey of New York lectured on public health law at the Harvard University School of Public Health on December 7 and 9, and will deliver lectures on the same subject at the Massachusetts Institute of Technology the middle of January.

A CONFESSED ABORTIONIST — Dr. James P. Nolan is reported to have pleaded guilty of attempting to perform an abortion. The patient died. The two accessories also pleaded guilty. Nolan split fees with one of the accessories. He received fifty and the woman who arranged for the operation kept twenty-five dollars.

Nolan evidently has no better opinion of himself than is the case with the average small criminal. Perhaps competition with others of his class has made it necessary to cut prices and split fees.

NOTICES

UNITED STATES CIVIL SERVICE EXAMINATION

Social Worker (Psychiatric)

Applications for social worker (psychiatric) must be on file at Washington, D. C., not later than January 18, 1927. The examination is to fill vacancies in the Veterans' Bureau, and in positions requiring similar qualifications throughout the United States.

The entrance salary is $1,860 a year.

The duties will be to investigate history and environmental conditions of patients; to analyze and submit data to the physician.

Full information and application blanks may be obtained from the United States Civil Service Commission, Washington, D. C., or the secretary of the Board of United States Civil Service Examiners at the postoffice or custom house in any city.

CORRECTION OF DATES

In the report of the meeting of the Boston Medical History Club on page 1178 of the issue of Dec. 16, 1926, four dates are given incorrectly. For 1837, 1838, 1840 and 1875 one should read 1537, 1538, 1540 and 1575.

FREE PUBLIC LECTURES ON MEDICAL SUBJECTS

The Faculty of Medicine of Harvard University offers a course of free public lectures on medical subjects, to be given at the Medical School, Longwood Avenue, Boston, on Sunday afternoons, beginning January 9 and ending March 27, 1927. The lectures will begin at four o'clock and the doors will be closed at five minutes past the hour. No tickets are required.

SUNDAY AFTERNOONS AT FOUR O'CLOCK

January 9—Dr. Richard M. Smith, Healthy Children.

January 16—Dr. Myrtelle M. Canavan, The Public Health Aspects of Venereal Diseases. (To Women only.)

January 23—Dr. Harry C. Solomon, Alcoholism, Syphilis and some other Conditions as Causes of Mental Disease.

January 30—Dr. Lawrence T. Fairhall, The Dangers of Overweight.

February 6—Dr. Joseph C. Aub, The Internal Secretions.

February 13—Dr. J. Dellinger Barney, Some Causes of Bladder Trouble. (To Men only.)

February 20—Dr. Douglas A. Thom, The Problem Child.

February 27—Dr. George H. Wright, How the Nerves, Eyes, Nose, and Throat may be affected by the Teeth.

March 6—Dr. William H. Robey, The Meaning of Blood Pressure.

March 13—Dr. Edwin T. Wyman, Fresh Air, Sunlight, and Vitamines.

March 20—Dr. Percy G. Stiles, Something about Dreams.

March 27—Dr. Shields Warren, Cancer and New Growths.

COURSE OF SIX LECTURES ON PREVENTIVE MEDICINE

SECOND SERIES, 1926-27

This course is offered by the Public Health Committee, Boston Section, Council of Jewish Women.

Place—Community Centre of Temple Ohabei Shalom, Beacon and Marshall Streets, Brookline. Time—3 p. m.

COURSE IN PREVENTIVE MEDICINE

Obesity—Monday, Jan. 17 (Open Council Meeting)—Lantern slides—Allan W. Rowe, Ph.D., Chief of Research Service, Evans Memorial Hospital.

High Blood Pressure and the Kidneys—Monday, Jan. 24—James P. O'Hare, M.D., Associate in Medicine, Peter Bent Brigham Hospital.

Gastro-Intestinal Disturbances—Monday, Jan. 31—Lantern slides—Percy B. Davidson, M.D., Thorndike Laboratory, Boston City Hospital.

Arthritis or so-called *Rheumatism*—Monday, Feb. 7—Louis M. Spear, M.D., Physician-in-Chief, Robert Brigham Hospital.

The Status of Cancer Research—Monday,

Feb. 14—Harry Friedman, M.D., Chief of Roentgenology Service, Boston Dispensary.

The Glands of Internal Secretion—Monday, Feb. 28 (Open Meeting)—Joseph Aub, M.D., Associate Physician, Massachusetts General Hospital.

There will be a fifteen minute question period after each lecture and literature will be on hand for distribution or sale.

The public is invited to the first and last lecture without charge.

Price—Regular course tickets, $2; single admission, 75 cents. Worker's course tickets, $1; single admission, 50 cents.

Subscriptions will be received by Mrs. Bernard D. Feinberg, 104 Summit Ave., Brookline.

REPORTS AND NOTICES OF MEETINGS

The annual meeting of the New England Association for Physical Therapeutics will be held on the premises of the Reconstruction Clinic, 366 Commonwealth Ave., Boston, on January 19, 1927.

Clinical demonstrations for application of Physical Therapeutic Measures will be held at the New England Sanatorium from two to four P. M., when luncheon to members will be served.

Clinical cases and demonstration will be presented at the Reconstruction Clinic from seven to eight-thirty P. M., preceeding the meeting and election of officers.

The meeting will be followed by a social hour and collation.

All physicians interested in this field of therapy are cordially invited to attend these clinics, which are the beginning of a regular series of clinical demonstrations to be given by the Association at regular meetings throughout the year, dates to be announced.

Signed: B. J. Manoogian, *Secretary*.

THE LAWRENCE MEDICAL CLUB

The monthly meeting of the club was held Monday evening, Nov. 22 with Harry H. Nevers, M.D., 246 Andover St., Lawrence. Chairman for the evening, John Parr, M.D., of Methuen. Subject: Medical Liability Insurance, Mr. Clarence T. MacDonald, Boston. Organized at eight-thirty o'clock.

The speaker explained the physicians' liability insurance as issued by his company and as approved by the Massachusetts Medical Society, and expressed the desire that a larger per cent of its fellows should be thus protected.

In the discussion the sentiment, as expressed by the audience, was that greater publicity by the insurance company and its agents would accomplish this end.

MEETING OF THE MASSACHUSETTS PSYCHIATRIC SOCIETY

A meeting of the Massachusetts Psychiatric Society was held at the Boston Psychopathic Hospital December 14, 1926. About sixty members and guests were present.

Dr. Percival Bailey spoke on "Newer Knowledge of the Neuroglia," and Dr. James V. Ayer on "Spinal Fluid Dynamics." Both talks were generously illustrated with lantern slides.

Winfred Overholser, *Sec.*,
Mass. Psychiatric Society.

HARVARD MEDICAL SOCIETY HOLDS SPECIAL MEETING

A special meeting of the Harvard Medical Society was held Tuesday evening, November 30, 1926, at the Peter Brent Brigham Hospital. The program for the meeting was a demonstration of cases, followed by a lecture by Dr. August Wimmer, Professor of Psychiatry at the University of Copenhagen; on "Epilepsy and Chronic Epidemic Encephalitis."

The first case was presented by Dr. Fulton of the hospital, which was one of palpitation of about three months' duration with no past history or family history. The trouble began as a very severe attack of pain in the chest, which has recurred along with a burning sensation underneath the sternum and a great deal of palpitation at irregular intervals. Physical examination showed a pulse of Corrigan Type, and an elevated blood pressure of 195 systolic over 60 diastolic. No evidence of infiltration of the arch and descending portion of the aorta, but a large shadow of the aorta, with considerable congestion of the lungs were shown by X-Ray. With the hand on the patient's chest a marked impulse was felt—and also a thrill extending from the sternum over the right side of the chest back to the vertebral column, this thrill being more commonly present during expiration than inspiration. A loud diastolic murmur was present. The patient had also a general heaving of the cardiac impulse over the whole of the pericardium with marked bulging. The other sounds of the heart indicated an aortic insufficiency.

Discussion of the case by Drs. Frothingham and Cushing followed with a diagnosis of insufficiency of the aortic valves, with other vascular complications.

The next case was presented by Dr. Bird of the Hospital Staff. The patient had an operation for duodenal ulcer in 1921, consisting of a transection of the stomach and a posterior

gastroenterostomy. Two years later a post-operative ventral hernia existed at the upper end of which was a small nodule which was then thought to be an infiltration or perhaps fibrosis around a stitch which had been left. Four months ago the case entered the hospital with upper abdomen symptoms, these being of a "popping or jumping" nature. This came on in spasms, usually six to eight impulses accompanied with protrusion of the abdomen. Physical examination showed that when the post-operative hernia was manipulated (or the nodule under the skin was touched) the patient would have spasms. Since the nodule seemed to move on a hinge from the lower end of the sternum a provisional diagnosis of fracture of the Xiphoid process was made. Operation: the mass was removed and a repair of the ventral hernia made in the usual fashion by overlapping. Since the operation the jump above mentioned, accompanied with spasm in the upper abdomen has occurred only once.

The case was diagnosed following discussion as diaphragmatic spasm. Such spasms have been found in cases having a brachial plexus injury, in cases of cervical rib and in cases of rupture of the stomach, especially by perforating ulcers which have extended to the diaphragm.

Dr. E. W. Taylor then introduced the speaker of the meeting, Professor August Wimmer. Dr. Wimmer has written on varied subjects from the psychoses to all types of neurological disturbances. He stands pre-eminent in the investigation of various disturbances of the brain, and especially of encephalitis and epilepsy. Dr Wimmer in introducing his subject praised th excellent and extensive work being done in this country on encephalitis.

Dr. Wimmer explained that in chronic epidemic encephalitis or epilepsy, we are dealing, so far as now known, with the sequelae of influenza infection of the brain. That the condition may be regarded as an inflamed condition of the brain, that is, a chronic progressive inflammatory disease. That, therefore, the form of epilepsy following so-called influenza is a quite different condition from the form of epilepsy in those who have a family history of epilepsy, or those who have developed epilepsy following trauma. He pointed out that epidemic encephalitis may follow immediately upon the initial attack of Spanish Disease or Influenza; or years may elapse before epileptic seizures appear. The mere fact that the patient has suffered from Spanish Disease does not justify a diagnosis of epidemic encephalitis because of the possibility of traumatism to the head and family and personal history of epileptic. Careful inquiry should be made into the course of previous Spanish Disease as well as to all illnesses which could possibly be related to the epileptic seizures for as expressed many years ago by Professor Pierre Marie, eminent French Neurologist, "Any infectious disease can be a causative factor in epilepsy," and this view Dr. Wimmer endorses.

Dr. Wimmer emphasized that care must be taken in making a diagnosis of chronic epidemic encephalitis. That this diagnosis must not be made in the case of children who, in childhood, have had fits, or in patients who have renal or heart conditions. He laid much emphasis upon spinal fluid tests, especially in cases where there is no history of previous Spanish Disease, but stated that no attention was paid to sugar reactions.

As to the cause of the epileptic fits in chronic epidemic encephalitis Dr. Wimmer volunteered the probability of a primary lesion in the base of the brain, explaining that in those cases which have come to autopsy, there has been demonstrated active pathological lesions. He pointed out definitely that the epileptic manifestations were always accompanied by changes in the central nervous system.

The epileptic fits so characteristically symptomatic of chronic epidemic encephalitis according to Dr. Wimmer's observations are of two types—those of but a minute's duration, which are of the pituitary type, that is, straining, loss of consciousness, falling down, restlessness, chronic spasms, involuntary passing of urine, foaming at the mouth and biting of the tongue —and those of several hours' duration, during which time the patient is unconscious, has involuntary movements of the feet and arms, but no foaming of the mouth and no biting of the tongue, but is exhausted upon regaining consciousness. Such symptoms as obesity, somnolence, headaches, involuntary movements and fever herald the epidemic form of epilepsy, and these symptoms are present throughout the disease.

As to the treatment of chronic epidemic encephalitis Dr. Wimmer stated there was little to be said. The only treatment possible in such cases is luminol, which is merely palliative.

A discussion of Dr. Wimmer's lecture followed and Dr. E. W. Taylor, acting chairman, in commenting upon the discussion and Dr. Wimmer's lecture stated, "The discussion has brought out more clearly than before the fact that we should regard epilepsy following influenza more as a symptom than a disease. Professor Wimmer has brought out quite well the fact that these epileptic seizures are only symptoms of a condition which he has described as encephalitis, rather than a distinct and definite disease."

SOCIETY MEETINGS

District Medical Societies

Essex North District Medical Society

Wednesday, January 5, 1927—Semi-annual meeting. Centre Church vestries, Main Street, Haverhill.

Wednesday, May 4, 1927—Annual meeting. Russell Hall, Young Men's Christian Association Building, 40 Lawrence Street, Lawrence.

Thursday, May 5, 1927—Censors meet for examination of candidates at Hotel Bartlett, 95 Main Street, Haverhill, at 2 P. M.

Essex South District Medical Society

Wednesday, January 5, 1927—Deer Cove Inn, Swampscott Dr. James S. Stone, "Differential Diagnosis of Acute Abdominal Conditions in Children." Discussion by Drs. O'Keefe of Lynn Nichols of Danvers and Walter Phippen of Salem, five minutes each.

Wednesday, February 2, 1927—Hawthorne Hotel, Salem. Dr H. H. Clute of the Lahey Clinic, "Differential Diagnosis and Treatment of Thyroid Disease." Discussion by Drs. Johnson of Beverly and Field of Salem, ten minutes each.

Wednesday, March 2, 1927—Lynn Hospital. Clinic, 5 P. M.; supper, 7 P. M. Dr. George Minot, "Pernicious Anemia, with Special Reference to Liver Diet." Discussion by Drs. Sargent of Salem and Reynolds of Danvers, ten minutes each.

Wednesday, April 6, 1927—Danvers State Hospital. Clinic, 5 P. M. Dr. Allan W. Rowe, Chief of Research Service at Evans Memorial, "The Differential Diagnosis of Endocrine Disorders." Followed by dinner. Discussion by Drs. Wood of Hathorne and Kline of Beverly, ten minutes each.

Thursday, May 5, 1927—Censors meet for examination of candidates at the Salem Hospital, 3:30 P. M.

Wednesday, May 11, 1927—Annual meeting. The Tavern Gloucester. Speaker and subject to be announced later.

Norfolk District Medical Society

Below are the proposed meetings of the Norfolk District for the remainder of the year. Minor changes may be made in case of necessity.

January 25, 1927—Peter Bent Brigham Hospital. Dr. Harvey Cushing. Time of meeting and subject to be announced.

March 1, 1927—Roxbury Masonic Temple, 8:15 P. M. Dr. Robert B. Greenough. To be devoted to a talk on cancer, with a résumé of the results of colloidal lead treatment.

March 29, 1927—Roxbury Masonic Temple, 8:15 P. M. Drs. F. S. Newell and F. J. Irving, "The Modern Treatment of the Eclampsias and Toxaemias of Pregnancy." If time permits—"The Modern Methods of Handling Prospective Caesarean Cases."

May 10, 1927—Annual meeting. Details of meeting to be announced.

Suffolk District Medical Society

Meetings of the Suffolk District Medical Society and the Boston Medical Library will be held at the Boston Medical Library, 8 The Fenway, Boston, at 8:15 P. M., as follows:

January 26, 1927—General meeting in association with the Boston Medical Library. "Medical Work at the Metropolitan Life Insurance Company," Dr. Augustus I. Knight, Medical Director, Metropolitan Life Insurance Company.

February 23, 1927—Surgical Section. "Clinic on Neurological Cases at the Peter Bent Brigham Hospital," Dr. Harvey Cushing.

March 30, 1927—Medical Section. Subject and speaker to be announced later.

April 27, 1927—Annual meeting. Election of officers. "Medical Education in the Orient and Occident," Dr. David L. Edsall, Dean, Harvard Medical School.

Notices of meetings must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.

BOOK REVIEWS

The Radcliffe Infirmary. By ALEXANDER GEORGE GIBSON. Oxford University Press, London: Humphrey Milford, 1926. vi + 316 pages.

The Radcliffe Infirmary is situated at Oxford and is one of the foundations left by Radcliffe in his will. The hospital has played an important part in medical development at Oxford and many well-known men have been associated with it as physicians or surgeons. Sir William Osler always took an interest in this little hospital, making regular visits and stimulating work in all its departments. Dr. Gibson, who was a close friend of Osler's and who looked out for him during his last illness, has written a very thorough history of the institution. He gives many interesting reminiscences of Osler and the following clinical aphorisms by Osler have been recorded, so far as I know, in no other publication (p. 153):

"1. Abdominal pain in old people: first think of hernia, and when you have done that, think of it again.

2. There are three requisites of a perfect medicine: it should be coloured, have taste and be harmless.

3. Pressure paraplegia is always a painful paraplegia.

4. Anterior poliomyelitis might be called Mephibosheth's disease, who was lame from infancy. Since the days of Madam Saul nursemaids have been thought to cause it by dropping their charges.

5. A patient may live many years with complete blockage of any one of the large veins, superior vena cava, inferior cava, portal vein.

6. All the organs of the abdomen may occasionally be seen through the abdominal wall except perhaps the coccygeal gland.

7. Ascites without an obvious cause means an operation.

8. Paroxysmal cough often means pus in the chest. (This was illustrated very strikingly in Osler's last illness.)

9. One swallow does not make a summer, but one tophus is sufficient for the diagnosis of gout.

10. Patients with imaginary ailments or with many complaints without obvious cause he would refer to as omphalites or as having a lesion of the filum terminale or pineal body."

Plastic Surgery of the Head, Face and Neck. By H. LYONS HUNT, M.D., L.R.C.S. (Edin.) Published by Lea & Febiger.

A very good treatise on plastic procedures about the head, face and neck, containing the usually accepted operations for the common congenital and acquired defects. Considerable attempt is made to include the much talked about "face lifting operations" and dignify them by scientific names and along anatomical lines. A perfectly legitimate and worthy attitude well taken by the author and done in such a way as to give the minimum offense.

The chapters on the history of plastic surgery and on local anesthesia are particularly good. The volume contains many original ideas which demonstrate the author's long study of the subject and his large experience with plastic surgery. His almost total lack of post-operative infection is indicative of unusually excellent technique.